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THE ETIOLOGY AND PATHOGENESIS OF FUNNEL CHEST, PIGEON BREAST, AND RELATED DEFORMITIES OF THE ANTERIOR CHEST WALL

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DEFORMITIES of the anterior chest wall of congenital or developmental origin have been discussed in the literature for more than 350 years. Nearly all of the articles have dealt with depression deformities, the protrusion deformities having been largely ignored; and, until recent years, the material has been limited to very few cases. Larger series of cases now being reported continue to be concerned with the depression deformities and little or no attempt has been made to classify these. A study of the anterior chest wall deformities of congenital or developmental origin indicates that, from the point of view of etiology and pathogenesis, the depression deformities and the protrusion deformities have much in common and it is with this phase of the subject that this paper deals. The material upon which this is based consists of personal experience with 161 operations in 150 cases (122 depression deformities and 28 protrusion deformities); a considerable number of cases which, for various reasons, did not come to operation; and 6 autopsies seen at Bellevue Hospital through the courtesy of the Department of Pathology.

These deformities fall into three groups, two of which seem to be exact opposites and the third to be a heterogeneous group of cases which have characteristics of both. In the first group are the depression deformities or funnel chests (pectus excavatum). In the second group are the protrusion deformities wherein are the pigeon breasts (pectus carinatum) and various other deformities characterized by protrusion. The third group consists of cases which have physical characteristics of both.

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To prevent ambiguity, funnel chest is defined as a funnel-shaped or conical depression of the anterior chest wall in which the sternum and attached costal cartilages curve backward, the xiphoid is at the apex of the funnel, *and the distance between xiphoid and vertebral column is abnormally short.* These deformities may be symmetrical or asymmetrical; and they may be present at birth or develop later as the child grows, particularly during the rapid growth associated with puberty.

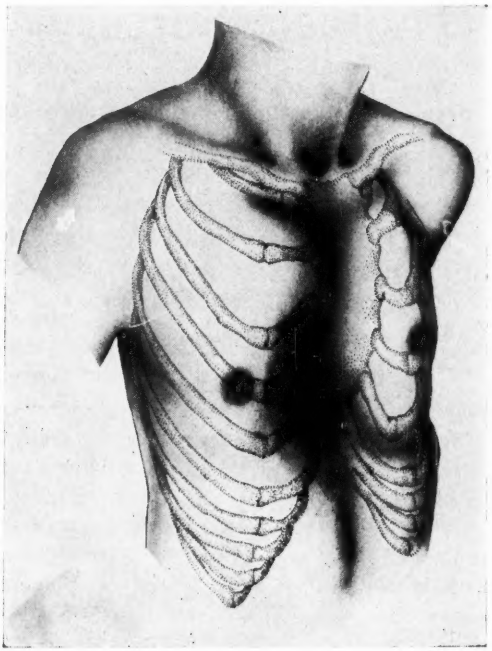

Fig. 1.—Funnel chest deformity of anterior chest wall.

Symmetrical deformities have the same amount of chest wall depression on each side of the midline and there is a transverse groove resembling the Harrison's groove of rickets (Fig. 1). The anteroposterior measurements of the chest are decreased to a greater or less extent thereby producing a caudad obliquity of the anterior ribs which is strikingly apparent on x-ray examination. The heart is displaced into the left chest. In young children, the abdomen has the appearance of a typical pot belly which is produced by the thoracic depression.

The asymmetrical deformity has the xiphoid in the deepest part of the depression as in the symmetrical type but the lateral portion of the deformity is more marked on one side than the other, usually the right. The other features are present but the obliquity of the ribs is greater on the more depressed side.

A characteristic of funnel chest in an infant or child is retraction of the xiphoid during inspiration. This paradoxic motion is almost never associated with dyspnea. When dyspnea is present, the other characteristics of funnel chest are usually absent because the retraction of the xiphoid is the result of the dyspnea and not the cause of it.

In the protrusion type of deformity, the protrusion may be in the midline and involve the sternum or it may be lateral to the sternum and correspond to the costochondral junctions, or in the costal arch. In the midline deformities the sternum may arch forward producing the shape of a pigeon breast (Fig. 2); or it may protrude in an oblique fashion with the xiphoid the most prominent part (Fig. 3). When the protrusion is lateral to the sternum it involves the costochondral rib junctions or the costal arch, which then flares (Fig. 4). The flaring costal arch does not necessarily appear on the same side

Fig. 2.—Arcuate type of pigeon breast in 6-year-old girl.

as the lateral deformity and may be on both sides. A flaring costal arch with the arcuate protrusion deformity simulates funnel chest (Fig. 5). However, there is adequate distance between xiphoid and vertebrae. The protrusion deformities may be present at birth but usually do not appear until the child has had a chance to grow, often not until adolescence. The deformity then seems to progress with growth.

Between the depression deformities and the protrusion deformities there is an indefinite group which seems to combine characteristics of both. The lower end of the sternum may be retracted while the upper portion may protrude. There may be an asymmetrical funnel chest with the more prominent side protruding like a lateral protrusion deformity. Occasionally a protrusion in early childhood or infancy may end up as a funnel chest after puberty. The group is, therefore, a heterogeneous mixture but the dominant features of each case tend to place it in one of the two main groups.

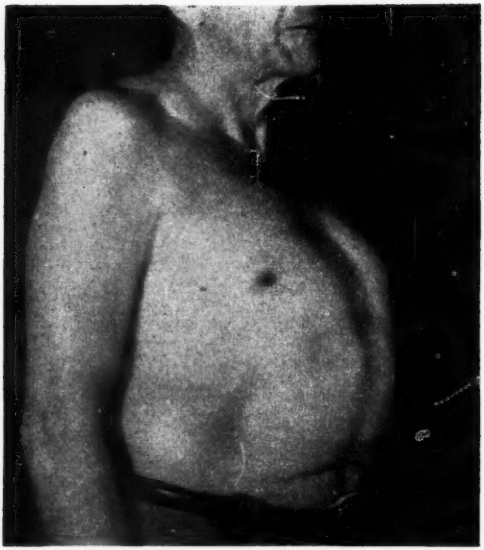

Fig. 3.—Oblique type of pigeon breast in a man 58 years of age. His twin sister has a similar deformity and her son has a funnel chest.

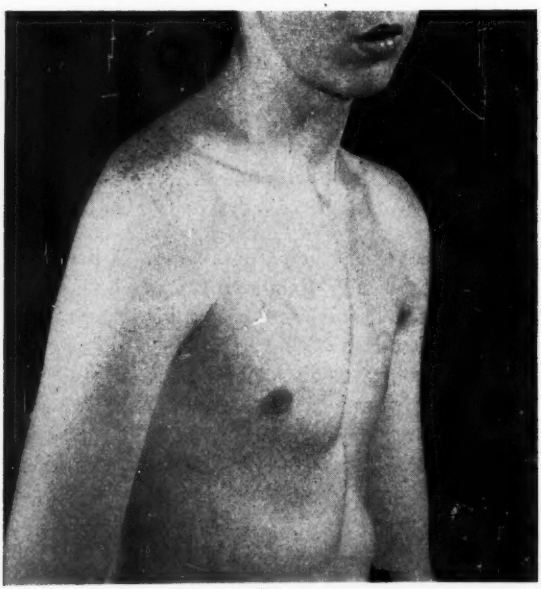

Fig. 4.—Lateral type of protrusion in a 14-year-old boy.

Heredity is mentioned as an etiological factor in funnel chest by nearly every writer on the subject. This factor is also noted in some of the few articles on protrusion deformities. Little has been said, however, of the rather striking fact that both types of deformity can appear in the same family. In the series on which this paper is based, there are numerous instances where one sibling has a funnel chest and another a pigeon breast; or where a parent, an uncle, or an aunt has a pigeon breast and the patient a funnel chest or vice versa. Tilney[2] tells of a pair of dissimilar twins, one with a funnel chest and the other with a pigeon breast. The hereditary factor, therefore, seems to apply to anterior chest wall deformities in general and not specifically to funnel chest or pigeon breast.

Fig. 5.—Arcuate pigeon breast deformity with flare of the costal arch previously reported[1] as funnel chest.

The anatomy of the thorax and its function as a bellows need not be discussed here except for two features. The first is the substernal ligament which was described by Brown[3] as an extension of the linea alba and the posterior rectus sheath up onto the posterior periosteum of the sternum with which it is intimately fused. Its dynamics are entirely those of the attached muscles.

The second is the diaphragm with its central tendon which is domed when relaxed at the end of expiration and flattened at the end of inspiration. It is an integral part of the chest bellows mechanism and essential in the production of an effective cough.

In a previous paper,[4] it was pointed out that the midline protrusion deformities are caused by proportionate overgrowth of paired ribs. If the overgrowth takes place in the upper ribs they will push the sternum forward as an arch (Fig. 6) and, in so doing, these ribs will buckle at their weakest points, usually the costochondral junctions, and the costal cartilages will slant forward to the elevated sternum. The formation of the arch is aided by the diaphragm which holds the xiphoid in place. If the ribs attached to the lower part of the sternum overgrow, the sternum is protruded in an oblique fashion (Fig. 7). If the diaphragm does not enlarge proportionately with the ribs, the protrusion is made at the expense of the dome of the diaphragm.

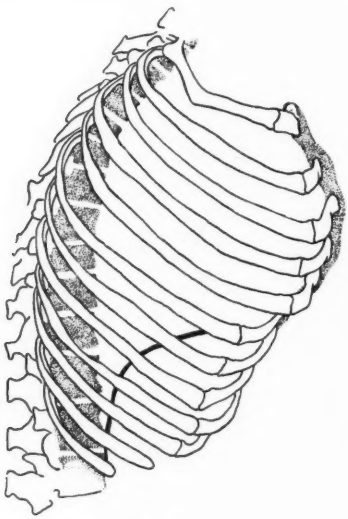

Fig. 6.

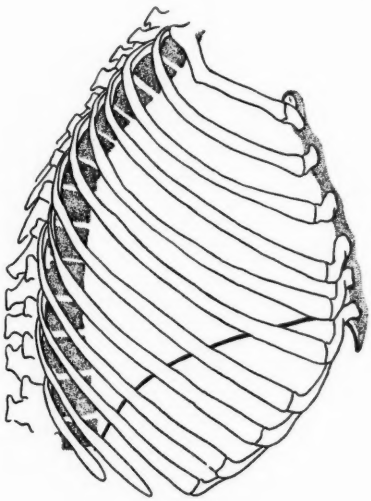

Fig. 7.

Fig. 6.—Arcuate type of protrusion deformity produced by proportionate overgrowth of second, third, and fourth pairs of ribs.

Fig. 7.—Oblique type of protrusion deformity produced by proportionate overgrowth of pairs of ribs attached to lower portion of sternum.

If the ribs above the level of the xiphoid are disproportionately longer on one side only, the ribs will again buckle at the costochondral junctions, but in this case the costochondral junctions will form a protruding ridge lateral to the sternum and their costal cartilages will slant backward to the sternum (Fig. 8). When overgrowth occurs in the ribs below the level of the xiphoid, the costal arch will flare, the diaphragm acting in the same manner on the costal arch that the sternum does on the upper ribs in the arcuate form of sternal protrusion.

Thus, the diaphragm seems to play a role of varying importance in the formation of these deformities. Observations made in the autopsy room tend to support this theory. In the depression, or funnel chest deformities, the diaphragm plays a much more important role.

Funnel chest deformities have a much more extensive literature than the protrusion deformities and this dates back to 1600 when Bauhinus[5] described such a case. Ochsner and DeBakey,[6] in an article published in 1939, list 142 references and since that time many other articles have appeared. Many of the authors speculate on the causes of the deformity but only three of these need be given serious consideration. These are: (1) the pull of a diaphragm with a short central tendon; (2) abnormal growth of the ribs; and (3) obstruction in the respiratory tract above the carina.

Bauhinus[5] believed from his observation that a muscular pull was responsible for the deformity, and the only muscle that can thus retract the sternum is the diaphragm. Woillez,[7] in 1860, also believed that the diaphragm was responsible and predicated that a condition similar to Dupuytron's contracture of the palmar fascia existed in the central tendon. Brown,[3] in 1939, believed that a short central tendon of the diaphragm was responsible and, on this basis, established the principle of the successful surgical treatment of the condition.

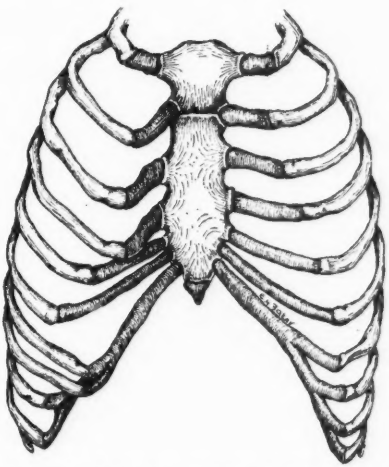

Fig. 8.—Protrusion deformity lateral to sternum produced by overgrowth of ribs 2, 3, and 4 on the right; and flare of right costal arch produced by overgrowth of ribs 7 and 8 on right.

Overgrowth of the ribs was mentioned by Flesch,[8] in 1873, as the cause of funnel chest, an idea which he ascribed to von Schiffer. More recently, Hausmann[9] also believes that this is the cause.

Obstruction in the respiratory tract was thought by Christian,[10] and some of the preceding editors of Osler's textbook, to be a factor in the production of funnel chest because, when this is present, the resulting dyspnea causes a marked retraction of the sternum which has the typical appearance of funnel chest. This will be considered later.

In the group of cases under consideration, observation indicates decidedly that a short central tendon of the diaphragm is the major factor in causing funnel chest. The salient features in support of this follow.

First: In infants and young children with funnel chest measurement of the anteroposterior diameter of the chest at the xiphoid show it to be retracted during inspiration often as much as 2 cm., while the same measurement in the midclavicular lines show the chest wall to move forward as much or more during inspiration. As children grow older the chest wall tends to lose some of its great resiliency and the retraction of the xiphoid decreases until, in adults, the xiphoid becomes fixed in a backward position while the forward inspiratory movement in the midclavicular line continues.

Second: When the patient is relaxed during sleep, the breathing is often noisy and the retraction greater. With greater relaxation under anesthesia, the retraction is exaggerated and the breathing may become quite labored. The use of an endotracheal tube eliminates the possibility of any obstruction above the carina. During the operation, the breathing difficulty increases as the costal cartilages are resected, often to such an extent that an assistant must exert traction on the xiphoid to restrain the paradoxic motion until the xiphoid is detached and the substernal ligament divided. When the diaphragm is thus released, respiration becomes so much easier and quieter that the anesthetist may become alarmed, fearing that it has ceased.

Third: Until a few years ago some depressions that were well corrected when the patient was discharged from the hospital tended to recur to a certain extent, especially after simple sternoplasty, and in a few instances reoperation was necessary. On the supposition that the regenerated seventh costal cartilage again allowed the diaphragm to exert its pull, the beds of the sixth and seventh cartilages and the intervening intercostal bundles were completely detached from the sternum along with the xiphoid. Since adopting this procedure, the corrections have been much better and the need for reoperation has not arisen.

Fourth: Examination at autopsy of 3 cases of funnel chest, one in a man 62 years of age, one in a woman somewhat younger, and the other in an infant of 2 years, showed that the central tendon of the diaphragm was actually much shortened. In the child, it measured 3 cm. and in the man of 62, only 1.5 cm. In each instance, the diaphragm presented a low dome for each leaf separated by the central tendon, instead of a much higher single dome as in the entire normal diaphragm, and strikingly demonstrates how seriously diaphragmatic respiration is compromised.

From the above, it is evident that the diaphragm is an essential factor in the production of funnel chest deformities, but, that overgrowth of the ribs may be a factor can also be demonstrated. It has just been mentioned that the deformity in some cases of funnel chest was absent or of minor extent in early childhood but developed as the child grew, particularly during the rapid growth of puberty. It is possible to explain this by the factor of rib growth.

In these cases during infancy it can be assumed that the central tendon of the diaphragm is moderately short or lacks the ability to grow. As the ribs lengthen the central tendon does not keep pace with them and the

diaphragm exerts an increasing pull on the lower end of the sternum, eventually producing a funnel chest with the same symptoms that obtain when the deformity is present at birth. Anatomically, the chest is not as much flattened nor is the caudad slant of the ribs as great as in the early cases.

Unequal growth of the ribs, as in the lateral protrusion deformities, can explain the asymmetrical funnel chest deformities. A short central tendon is present as in the symmetrical deformities but the ribs grow more on one side than on the other and the depression becomes deeper on the side where the ribs are shorter. As in the case of the protrusion deformities, the asymmetry is absent or minimal during infancy and increases with growth.

In the indefinite group of anterior chest wall deformities, the same factors obtain. A central tendon only slightly shortened in the presence of rib overgrowth can give the appearance of a funnel chest although the xiphoid does not have a paradoxic motion. Carrying this a step farther, there may be an arcuate type of pigeon breast with a definitely shortened central tendon and paradoxic respiration. Two such cases in this series had the symptoms of funnel chest but the arcuate protrusion of the sternum caused the short central tendon to pull the sixth costal cartilages behind the fifth so that, at operation, the fifth costal cartilage on each side had to be resected before the sixth could be approached.

A third case was essentially one of protrusion deformity with bilateral flares in the costal arch and an arcuate type of pigeon breast together with a bad round back posture. Release of the diaphragm with correction of the flares and elevation of the lower end of the sternum allowed the child to maintain an erect posture which was not possible before operation.

Another case was followed for five years as a protrusion deformity but, with the accelerated growth of puberty, respiratory difficulty and decreased exercise tolerance appeared and it was then noticed that the diaphragm was moving in a paradoxic manner. The radical operation for funnel chest relieved the symptoms.

Thus, it can be seen that the cases in this heterogeneous group present some clinical characteristics of both protrusion deformities and depression deformities. The symptoms, however, are produced by the dominant factor.

Pseudo-funnel chest is a name which can be given to another group of cases often confused with funnel chest but actually entirely different. The condition is noted at birth or shortly thereafter and is characterized by dyspnea and marked paradoxic motion of the xiphoid. The dyspnea may be the result of pneumonitis from the aspiration of amniotic fluid, a congenital heart lesion, obstruction in the respiratory tract or any other condition which can cause dyspnea in a newborn baby. Forced inspiration with a quick build-up of negative pressure produces the paradoxic motion and not a short central tendon of the diaphragm. The treatment is the treatment of the cause of the dyspnea, but traction on the xiphoid to restrict or prevent the paradoxic motion may bring dramatic relief.

SUMMARY AND CONCLUSIONS

Study of a considerable number of cases indicates that anterior chest wall deformities characterized by depression and those characterized by protrusion can be classified together.

Heredity is a most important etiological factor and either type of deformity can be inherited from the same ancestor.

Size and shape of the diaphragm and disproportionate growth of the ribs are the principal factors in the pathogenesis of these deformities.

If a short central tendon of the diaphragm is the dominant factor, funnel chest develops. If disproportionate overgrowth of the ribs is the dominant factor, one of the types of protrusion deformity will result. When both factors are present in the same individual the dominant deformity will be modified.

Pseudo-funnel chest is an inspiratory retraction of the anterior chest wall in an infant shortly after birth which is due to some pathologic condition in the cardiorespiratory system and not to the factors of true anterior chest wall deformities.

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THE INFLUENCE OF INTRA- AND EXTRATHORACIC OPERATIONS ON THE ELECTROCARDIOGRAM IN PATIENTS WITH PULMONARY TUBERCULOSIS

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THE effect of the various active methods of treatment of pulmonary tuberculosis on the electrocardiogram has been studied by many investigators, and the electrocardiographic changes caused by pneumothorax treatment, phrenic exairesis and, in later years, by thoracoplasty and pulmonary resection, have been described extensively.

Weinshell and associates[15] examined 45 patients and they arrived at the conclusion that practically all electrocardiographic alterations were due to changes in position of the heart. They studied this in pneumothorax, pneumoperitoneum, thoracoplasty, and pneumonectomy. Weiss[16] believed that great importance should be attributed to the position of the mediastinum. When the mediastinum was displaced to the left, he observed a more vertical position of the heart; he also ascribed the occurrence of negative T waves in the precordial leads to this displacement. A subclinical cor pulmonale, developing in the postoperative period, was also said to be a factor in the formation of these negative T waves. Thus, changes in the position of the heart, as well as myocardial abnormalities, might be the cause of postoperative electrocardiographic changes. To illustrate this, Weiss cited Biocca and his co-workers[3] who attributed late T changes after lobectomy and pneumonectomy to myocardial damage. During the first few postoperative days, Biocca observed changes in the S-T segments, which entirely resembled the abnormalities found in acute pericarditis.

Armen and Frank[1] likewise observed changes of the T waves, but they believed that these were caused by the presence of air between heart and chest wall. The electrocardiographic abnormalities occurred most frequently after a left pneumothorax treatment. In only 4 out of 68 patients did Hertzman and Mathisen[7] find postoperative electrocardiographic changes which, in their opinion, were not due to the altered position of the heart because these 4 patients showed an isoelectric T wave in the first standard lead.

The same abnormality of the T wave in the first lead was observed by Rauchwerger and Erskine.[12] These authors were unable to find a satisfactory explanation of this phenomenon. Hoppe[8] frequently observed a P pulmonale after pulmonary resection and he saw also transient S-T and T changes.

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The following pages deal with our personal observations of electrocardiographic changes in a large series of patients with pulmonary tuberculosis, treated by thoracoplasty or pulmonary resection.

METHODS OF EXAMINATION

In every electrocardiographic examination a great number of leads were taken, namely, the three standard leads, the three unipolar leads according to Goldberger, and the precordial leads according to Wilson from V_1 to V_6, inclusive.

The position of the heart was determined according to Wilson's criteria; in order to avoid micro-electrocardiography the semivertical and the semihorizontal positions were regarded as intermediate.

A preoperative electrocardiogram was made of more than 500 patients selected for operation; special attention was given to the position of the heart. In 444 patients of this group a postoperative electrocardiogram was recorded (at least six months after the operation); this electrocardiogram was compared with the preoperative one.

In a consecutive series of 113 patients of this group, one or more electrocardiograms were also made in the immediate postoperative period. In 28 consecutive patients of the latter group, the arterial oxygen saturation was determined simultaneously with the electrocardiogram. This was done by means of the hemoreflector of Brinkman.[17] The blood was taken from the brachial artery. To augment the accuracy of the determination, the point of maximal saturation as well as the point of maximal desaturation of each blood sample was also determined (Zijlstra[17]).

The presence or absence of pneumothorax was verified in 63 of the 113 patients mentioned by means of x-ray pictures made simultaneously.

RESULTS

There was a remarkably small number of patients in whom electrocardiographic abnormalities were found before the operation; only 12 of the 508 patients were to be regarded as having a pathologically changed electrocardiogram. In 10 patients, the T waves were abnormal; the standard leads, the unipolar as well as the precordial leads showing abnormalities in these waves. T_{III} was negative in all these 10 patients; in 5 of them T_{II} was isoelectric or negative. Also, in 5 patients the T_I was negative or isoelectric. In all patients, T_{aV_F} was pathologic, while 3 patients had a negative T wave in all precordial leads. Less severe T-wave changes in the precordial leads were present in the other patients. In 9 of these 10 patients, the T-wave abnormalities had improved after operation or had entirely disappeared; in one patient they remained unchanged. Two of these 12 patients showed a disturbance of conduction; one a prolonged P-R interval and the other an arborization block.

Table I lists the data of the position of the heart in the 508 patients and the distribution over the various age groups. According to this table, an intermediate position of the heart was found in 51 per cent of the cases, while

a horizontal position was observed in only 8 per cent. This should probably be attributed to the fact that the investigation dealt mainly with patients of the younger age groups.

TABLE I

POSITION OF THE HEART	10-19 YEARS	20-29 YEARS	30-39 YEARS	40 YEARS AND OLDER	TOTAL	PER-CENTAGE
Intermediate	9	62	44	25	140	28
Intermediate cwr*	10	33	38	16	97	19
Intermediate ccwr†	1	8	10	3	22	4
Vertical	18	38	25	18	99	20
Vertical cwr	7	33	30	11	81	16
Vertical ccwr	3	10	7	5	25	5
Horizontal	-	5	6	5	16	3
Horizontal cwr	-	8	8	12	28	5
Totals	48	197	168	95	508	100

*cwr = clockwise rotation.
†ccwr = counterclockwise rotation.

Comparison of the preoperative and postoperative electrocardiograms of 444 patients proved that there was still a manifest change in position in 94 cases six months after the operation (that is, in 21 per cent of cases).

Table II shows the distribution over the various operations.

TABLE II

TYPE OF OPERATION	TOTAL NUMBER	CHANGE IN THE POSITION OF THE HEART AFTER OPERATION	
		TOTAL	PER CENT
Thoracoplasty	60 } 79	11 } 14	18
(Schede modification)	19 }	3 }	
Pleurectomy	17	3	18
Segmental resection	233	45	19
Lobectomy	78	16	21
Pneumonectomy	37	16	43
Totals	444	94	21
Right-sided operations	208	37	18
Left-sided operations	236	57	24

It is evident that the frequency of change of position was significantly higher after pneumonectomy than after thoracoplasty, segmental resection, or lobectomy. The latter groups show only slight mutual differences. An operation on the left hemithorax led to a somewhat higher percentage of changes of position than an operation on the right side, but this difference is not statistically significant. After a left pneumonectomy there was a tendency to a more horizontal position of the heart. The same was observed after left and right lobectomy. In the other operations, no particular tendency could be distinguished.

Directly after the operation the electrocardiogram usually showed an increase of the frequency combined with a diminution of the amplitude of the QRS complex.

Thirty-five of the 113 patients, who were electrocardiographically examined immediately postoperatively, also had other abnormalities that were to be regarded as pathologic. These abnormalities were always located in the S-T

segments and the T waves. In 5 patients, elevated S-T segments were found, namely, in Leads I, II, aV_L and V_6 (Fig. 1, *D*). The other patients only showed changes in the T waves; the most frequent observation was negativity of the T waves in all precordial leads. This was the case in 21 patients (Fig. 1, *A*, *B*, and *F*).

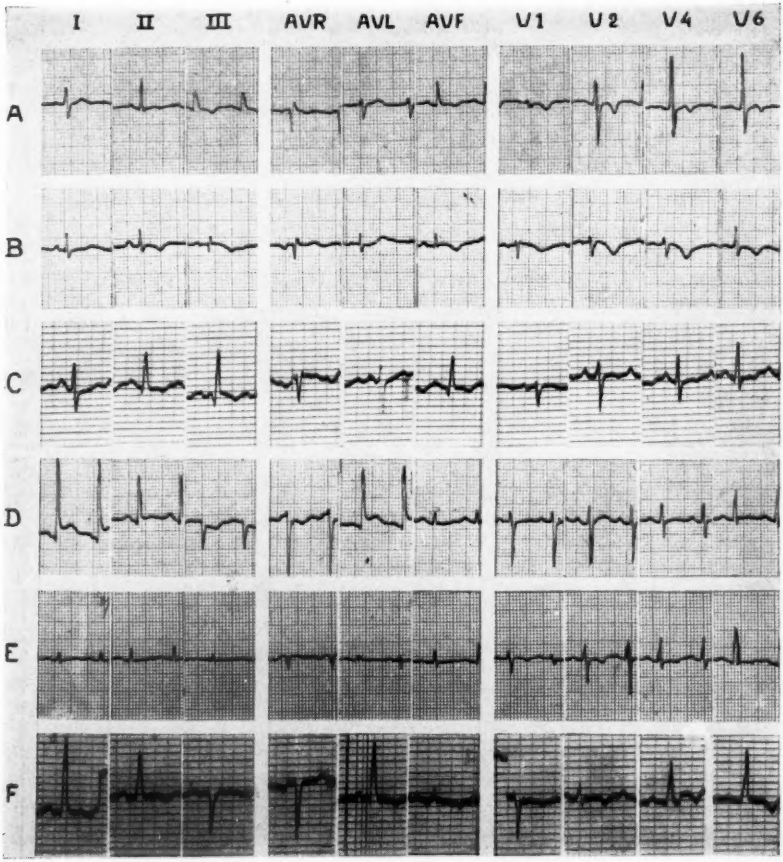

Fig. 1.—Electrocardiograms of 6 patients recorded in the first two weeks after operation.
A, right-sided lobectomy two days after operation.
B, seven days after left-sided pneumonectomy.
C, two days after right-sided lobectomy.
D, two days after left pneumonectomy (note S-T deviation).
E, eleven days after left pneumonectomy (in this case pericardial puncture was necessary).
F, seven days after left pneumonectomy.

In some other patients not all precordial leads were pathologically changed but only part of them, especially the right precordial ones. The T wave in aV_F was isoelectric or negative in nearly all patients. T in aV_L was practically

always positive. Every one of these 35 patients showed also abnormalities in the T waves in the standard leads. The T_{III} was always isoelectric or negative; in 5 cases the T_I was abnormal (Fig. 1, *F*).

These S-T and T-wave changes gradually disappeared in most patients, sometimes as early as after a week, in other patients after some weeks or months (Fig. 2). Only in one case did the changes of the T waves persist for longer than six months.

Table III shows the relationship between the occurrence of the S-T and T-wave changes and the nature of the operation. It is a striking feature that the number of cases with abnormalities of the T waves is relatively greater in pneumonectomies than after the other operations. On the other hand, the above-mentioned abnormalities do not arise at all after thoracoplasty.

TABLE III

TYPE OF OPERATION	TOTAL NUMBER OF PATIENTS	S-T AND T-WAVE CHANGES	
		NUMBER	PER CENT
Thoracoplasty	21	0	0
Segmental resection	53	17	32
Lobectomy	19	5	26
Pneumonectomy	20	13	65
Total	113	35	31

We also studied whether there was any relationship between these changes in the T waves and the postoperative arterial oxygen saturation. As no negative T waves were observed after thoracoplasty, we separately considered the thoracoplasty cases in the consecutive series of 28 patients in whom the arterial oxygen saturation was determined during the first postoperative weeks; 8 patients in total.

Table IV contains the data obtained in these 8 patients. It was found that the arterial oxygen saturation can fall to low levels after thoracoplasty. This severe and long-lasting undersaturation was especially observed in the

TABLE IV. THORACOPLASTY CASES

CASE NUMBER	OXYGEN SATURATION (%)		S-T- AND T-WAVE CHANGES
	BEFORE OPERATION	AFTER OPERATION	
1	95	94½	Absent
2	96	91	Absent
3	-	93½	Absent
4	92½	90	Absent
5	96	84	Absent
6	96	94	Absent
7	91	89	Absent
8	-	75	Absent

patients in whom thoracoplasty had resulted in a pendular movement of the anterior chest wall, refractory to corrective measures. It must be pointed out that in each instance the lowest oxygen saturations of the first postoperative weeks are listed in the tables. The electrocardiogram and the oxygen saturation were frequently determined, so long as the latter value had not reached

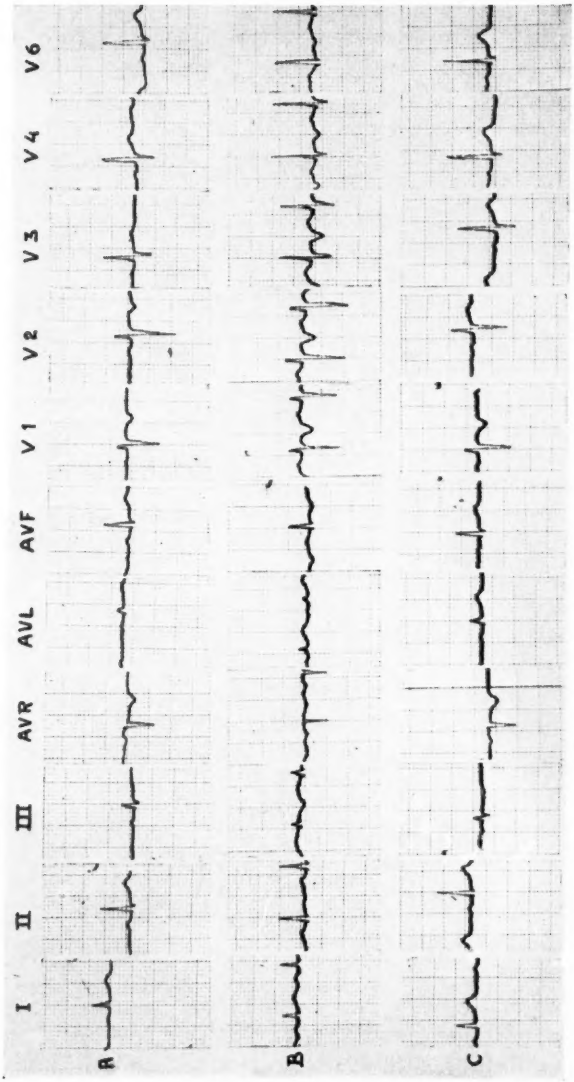

Fig. 2.—Electrocardiograms of a patient, 30 years of age. *A*, before operation, *B*, three days after segmental resection in the right upper lobe. *C*, seven months afterward.

preoperative levels. This was usually the case in the first postoperative week; Patients 5 and 8 from Table IV, however, needed more than a fortnight before the oxygen saturation had become normal (Maier and Cournand[10]).

It is evident that even a markedly lowered oxygen saturation was not accompanied by electrocardiographic changes as described above. At most, some flattening of the T waves in the first standard lead and in V_6 was observed, probably as an anoxemia reaction.

Table V gives the same data of the 20 other patients, who underwent some form of resection therapy. In these patients also the arterial oxygen saturation sometimes fell considerably in the postoperative period and in a number of them the S-T and T-wave changes arose. On the other hand, these abnormalities were also observed in the patients in whom the oxygen saturation did not fall at all after the operation. The table shows that there does not exist any relationship between the occurrence of the electrocardiographic changes and the arterial oxygen saturation.

TABLE V. RESECTION CASES

CASE NUMBER	OXYGEN SATURATION (%)		S-T- AND T-WAVE CHANGES
	BEFORE OPERATION	AFTER OPERATION	
1	92	79	Absent
2	–	94½	Absent
3	96	90	Absent
4	95	90	Absent
5	95	91	Absent
6	96	95	Absent
7	95	61	Absent
8	95	86	Absent
9	96	93	Absent
10	–	58	Only T_{V6} negative
11	96	88	Present
12	97	90	Present
13	96	93	Present
14	96	90	Present
15	96	90	Present
16	96	95	Present
17	–	92	Present
18	98	94	Present
19	96	94	Present
20	94½	90	Present

On the basis of the radiologic findings of 63 cases, we finally studied whether there was a correlation between the electrocardiographic abnormalities and the presence of air in the thoracic cavity.

Thirty-two patients had undergone a left-sided resection; in 16 of them the T-wave changes developed in the first week, while only 3 of them had a pneumothorax at the same time. In 6 other patients, however, a pneumothorax was present without any changes on the electrocardiogram. The same ratio was observed in the operations on the right side. It was impossible to demonstrate a relationship between the presence of air in the pleural cavity and the occurrence of electrocardiographic changes.

DISCUSSION

Only in 12 of the 508 patients examined did an abnormal electrocardiogram exist before the operation. This is quite consistent with the investigation carried out by Harden and his associates[6] who found only one abnormal electrocardiogram in 95 patients. Björkman,[4] however, arrived at quite different figures. These differences are probably due to varying opinions on what should be considered normal and abnormal in the electrocardiogram. We must remark that we did not regard an isolated negative T wave in Lead III or aV_F as pathologic. Changes in the position of the heart are generally described after intrathoracic operations (Harden,[6] Weinshell,[15] Hertzman[7]). A distinction must be made between the transient changes of the position of the heart, as observed shortly after the operation, and the final changes found later, for example, after six months. The frequent occurrence of a permanent change in the position after a left pneumonectomy is an especially striking feature.

The change of the position as such may give rise to a flattening or turning negative of the T waves in various leads (Goldberger[18]). An electrocardiogram recorded in the first week following operation may show quite different changes, localized in the T waves and S-T segments. We observed these changes in 35 of the 113 patients examined for this. Negative T waves were found in all precordial leads, while a negative T_I and T_{II} were also observed. In these cases we could speak of massive T-wave changes. These abnormalities were usually of short duration, lasting only for some weeks. T-wave changes have also been described in the literature, but only a few investigators have carried out electrocardiographic investigations during the first postoperative week (for example, Hoppe[8]). It is only possible to get information about these electrocardiographic abnormalities by making an electrocardiogram as early as possible after the operation.

Opinions vary on the cause of the T-wave changes after resection therapy for tuberculosis.

We cannot confirm Weiss's[6] opinion that a displacement of the mediastinum to the left may be an etiological factor. This displacement was observed several times in our patients with left pneumonectomies. The negative T waves usually disappeared rapidly, while the displacement of the mediastinum persisted or even became worse. In the only patient with the persistent negative T waves, the mediastinum was not displaced to the left.

The presence of air between the heart and the chest wall has also been mentioned as a cause of the T-wave changes (Armen[1]). We were unable to find any indications of this.

Lesions of the myocardium (''myocardial damage'') due to hypoxia have also been considered responsible (Katz,[9] Rahman[11]). We did not find any correlation between the occurrence of electrocardiographic changes and the arterial undersaturation.

Refrigeration during the operation (Rahman[11]) does not seem to be of importance in this respect.

Digitalis preparations may cause manifest S-T and T-wave changes, which may to some extent simulate the abnormalities described by us. We did not treat our patients with any of these preparations, so that this causative factor can be ruled out.

According to some investigators (Weiss[16]), overloading of the right heart after pulmonary resection might give rise to changes of the electrocardiogram. We did not believe that this is probable, in view of the investigations of Sluiter[13] and Schweitzer[14] who did not observe such T-wave changes even in the case of a completely developed cor pulmonale.

The great similarity to the picture of acute pericarditis was a striking feature in all the cases with extensive T-wave and S-T changes in the postoperative electrocardiogram. Fig. 1, *D* is very illustrative in this respect, as the characteristic S-T elevations are present. This similarity was also observed by the Italian investigator di Maria (cited by Harden[6]). We are, therefore, of the opinion that the electrocardiographic abnormalities found by us, in patients who had undergone pulmonary resection, were attributable to changes of the pericardium. In one of our patients, a pericardial puncture was required, as the circulation had been seriously impeded due to accumulation of fluid in the pericardium (electrocardiogram of this patient: Fig. 1, *E*. This electrocardiogram shows a somewhat later stage in which the S-T elevations are diminished and the changes in the T waves appear). Bérard and Jacquemet[2] observed an increase of fluid in the pericardium in 4 out of 500 cases of pulmonary resection (only after pneumonectomy). They did not carry out electrocardiographic examinations, however.

During the first postoperative period, electrocardiographic changes such as those just described after pulmonary operations are also observed after certain heart operations; commissurotomy of the mitral valve in mitral stenosis, and valvulotomy of the pulmonary valves in cases of pulmonary stenosis. In these cardiac operations, the pericardium is opened and it is conceivable that a condition of irritation of the two pericardial leaves may develop. On several occasions, Campbell and Brock[5] had to perform pericardial puncture after a valvulotomy for pulmonary stenosis. Analogous to this it seems to us that in a number of cases of pulmonary tuberculosis a transient, usually symptomless, pericardial irritation may be caused by intrathoracic operations with manipulations in the neighborhood of the pericardium. This irritation many give rise to a typical electrocardiographic picture, which proves to be reversible in most cases. We were strengthened in this opinion by the absence of electrocardiographic abnormalities in the S-T and T waves after thoracoplasties. Pericardial aspiration would appear to be indicated in cases in which this electrocardiographic picture is observed in combination with a weak pulse and other signs of fluid accumulation in the pericardium.

SUMMARY

1. The electrocardiographic findings in more than 500 patients with pulmonary tuberculosis were studied. Special attention was given to the position of the heart in connection with age. In only 12 patients a pathologic electrocardiogram was found; the abnormalities were mainly located in the T waves.

2. In 444 patients, the electrocardiogram, recorded at least six months after the operation (thoracoplasty and pulmonary resection), was compared with the preoperative electrocardiogram. A change in position of the heart was observed in 94 patients. This change of cardiac position was most frequent after left pneumonectomy.

3. Repeated electrocardiographic examinations were carried out during the first postoperative weeks in 113 consecutive patients from this series. Thirty-five of them showed extensive changes in the S-T and T waves which developed soon after the operation and persisted during a number of weeks but disappeared in most cases within six months after the operation.

4. The cause of these electrocardiographic changes in the postoperative period is discussed. Based on the marked similarity of the electrocardiographic picture with that of pericarditis and on the strength of clinical observations, it is considered probable that the abnormalities found are caused by pericardial irritation.

All the operations mentioned were performed by Prof. L. D. Eerland, M.D. (Director of the Surgical Clinic, University Hospital, Groningen, Holland).

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THE USE OF A PLASTIC SPONGE (IVALON) IN OPERATIVE PROCEDURES FOR PULMONARY TUBERCULOSIS

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THE use of space-taking devices has long been a part of the treatment of pulmonary tuberculosis. It is said that therapeutic injections of air into the thorax were advocated at the time of Hippocrates. Gilchrist,[1] in the middle of the eighteenth century, recommended pneumothorax; and Carson,[2] in 1819, described the pulmonary physiology with an amazing degree of accuracy and advised intrathoracic injections of air for "quiescence" of a diseased lung. Others made similar pleas for some type of collapse therapy of the lung with pneumothorax, but it remained for Forlanini[3] of Italy, in 1882, to describe an aseptic method and specific technique which made pneumothorax a standard of treatment for tuberculosis, and others later to make it a "must" for uncontrollable hemoptysis.

DeCerenville,[4] in 1885, described a method of anterior thoracoplasty allowing the cut ends of the ribs to come together and reducing the circumference of the chest to close tuberculous cavities. Others reported similar operations, as did Brauer[5] and Friedrich[6] who popularized massive rib resections in Europe in the early part of the twentieth century. Later, Wilms,[7] Sauerbruch,[8] and Alexander[9] developed the safe, conventional, staged thoracoplasty done today.

Tuffier,[10] in 1891, first described his "extrapleural pneumonolysis" consisting of stripping the pleura from the rib cage over the apex of the lung through an incision in the second interspace anteriorly and letting this space fill with air. In 1901, Sarfert[11] performed a similar procedure, using a gauze packing to maintain the depression of the pleura. Tuffier then reported on similar cases in which he filled the space with grafts of adipose tissue, omentum, and fibromas. Later he used refills of air and paraffin, though the latter filling had been reported first by Baer[12] in 1913. Many other filling substances have been reported: Lilienthal,[13] 1928, gum sheeting; Alexander,[14] 1930, autogenous grafts of pectoral muscles; Oughterson and Harvey,[15] rubber bags; Eloesser,[16] 1932, paraffin.

With the development of pulmonary resections in the treatment of pulmonary tuberculosis, there was an even greater stimulus for further search for space-taking devices. Since the introduction of plastics, various of these materials have been used for the purpose of filling spaces in the bony thorax in the treatment of tuberculosis. Lucite balls were first used by Wilson and Baker.[17] Later, the use of Polystan (polyethylene) sponges were reported by

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Bing and associates.[18] In 1951, Grindlay and Waugh[19] at Mayo Clinic reported the use of a formalinized polyvinyl alcohol sponge in various parts of the body, including the chest.

Conventional thoracoplasties, while having the advantage of being entirely nonirritating, have the marked disadvantages of requiring several stages and leaving large portions of chest flail, making for respiratory difficulties during

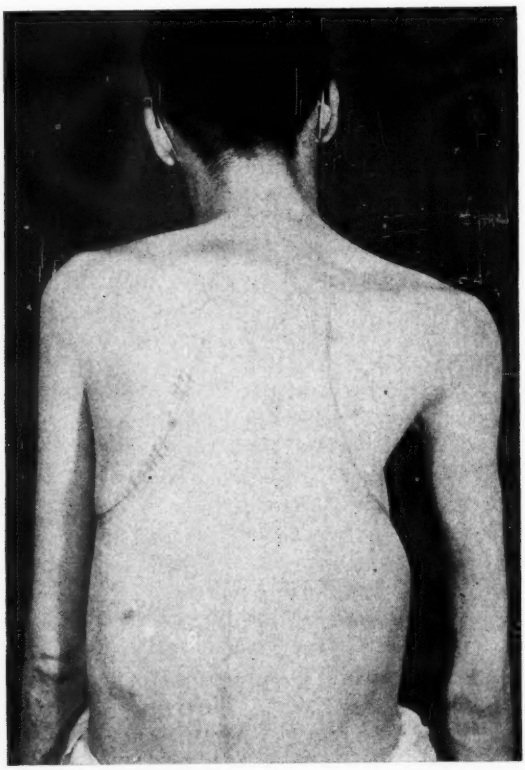

Fig. 1.—Photograph of M. H., 28-year-old white man, who had a conventional three-stage, 7-rib thoracoplasty on the right, a 7-rib plombage thoracoplasty on the left. Note the scoliosis, rib cage deformity, elevated shoulder and muscular atrophy on the right secondary to the conventional thoracoplasty.

the healing period. Unfortunately, in addition, they impair the breathing capacities, as well as being extremely disfiguring. Plombage, for either primary or secondary procedures, has the advantages of being a single procedure, of avoiding the flail chest, reducing the operative risk, obtaining better and more exact collapse, and reducing pain and loss of mobility of the adjacent arm. Equally as important is the preservation of pulmonary function. Watson and Gaensler[20] demonstrated a drop in pulmonary function of only about one-half as much with plombage thoracoplasty as with conventional

thoracoplasty, this work being done on patients who had Lucite balls used for the plombage thoracoplasty. This makes it feasible to operate unilaterally and bilaterally on poor-risk patients that could not tolerate the classic staged thoracoplasty (Fig. 1).

Of further importance is a factor that has not been sufficiently stressed. Economically, the plombage procedures offer a tremendous saving in many ways, especially in the presence of a backlog of patients. In a given time with a fixed personnel, more plombage procedures can be performed than conventional thoracoplasties or resections. Postoperatively, the patients do extremely well and require far less care. In addition, a shorter period of total and postoperative hospitalization is required for these patients.

MATERIAL USED AS PLOMBAGE

As just mentioned, many different types of material have been used for plombage and reported upon. Each has its advantages and disadvantages. Growing body tissues would seem to be the most desirable. However, the obvious disadvantages are that they are difficult to obtain, scant in quantity, and tend to atrophy. Inert foreign substances would, of course, seem to be the next most feasible. Ideally they should be: (1) stable, (2) chemically inactive, (3) compressible, (4) able to produce minimal foreign body response, (5) easily shaped, (6) light in weight, and (7) of such a nature that they might be incorporated into the body tissues. That such properties may be assigned to formalinized polyvinyl alcohol sponges was first demonstrated by Grindlay and Waugh,[19] and later confirmed by Gale and co-workers.[21] We have used this type sponge exclusively.

TYPES OF OPERATIONS AND INDICATIONS

At the Mississippi State Sanatorium, we have been using the Ivalon sponge for a period of four years. This report deals with the utilization of 150 sponges in the first 144 consecutive patients who have been followed from one to four years.

The plombage procedures are classified as "Primary" if used for collapse and as "Secondary" if used as space fillers following resection. The primary procedures were usually plombage thoracoplasties in which the sponge was placed subcostally, extraperiosteally as a permanent implant. In some cases, however, the sponge was placed extrapleurally after stripping the parietal pleura from the chest wall.

Secondary plombages were usually placed extrapleurally or extraperiosteally as a tailoring procedure in cases where a lobe or more pulmonary tissue was removed and the remaining lung did not readily fill the hemithorax. These plombages were preferably introduced as a part of the same operation wherein the resection was carried out, though some were done at a second operation. In a few cases, following pneumonectomies, the sponges were introduced intrapleurally after a minimum postoperative delay of three months.

Indications for the secondary space-filling procedures are essentially as outlined by Cotton and Paulsen[22] and may be briefly summarized as the prevention of overdistention if (1) the remaining lung will not easily fill the chest or if (2) extensive residual disease, particularly of the contralateral apex, makes any degree of overdistention undesirable. The indications for the primary plombage are somewhat more controversial even though, basically, they were set forth by Tuffier,[23] the originator of "extrapleural pneumonolysis" and have changed very little since that time. These are: (1) uncontrollable hemoptysis from an apical lesion, (2) fibrocavitary tuberculosis limited to the upper portion of the lung, (3) supple cavities less than 4 cm. in size, (4) lesions of tuberculosis which might well be resected but for a positive sputum with bacilli resistant to present antituberculous medications. In addition, (5) plombage thoracoplasties are preferred for unilateral and bilateral procedures in patients with markedly impaired respiratory reserves, and (6) where the distribution of the disease makes resection of all significant residuals impractical or impossible, but allows collapse of open cavitary apical lesions.

The primary extrapleural plombage has been used only in cases where there is some good evidence of healing of the disease prior to surgery and there is no evidence of cavitation within a centimeter of the periphery of the lung. With extreme care in the selection of patients, avoiding the pitfalls emphasized by Zimmerman and his associates,[24] the risks should be minimal, but at the same time the scope of this procedure will be severely limited.

OPERATIVE METHODS

Primary plombage thoracoplasties are performed much as originally outlined by Wilson and Baker.[17] The usual parascapular incision is made as for a conventional thoracoplasty and the inferior extent of the thoracoplasty is determined by the usual criteria for conventional thoracoplasty. Beginning with the lowest rib and continuing through the first, the periosteum is stripped only from the under surface of the rib, leaving the outer surface intact. The posterior muscle bundles are dissected free from the ribs subperiosteally by blunt dissection around to the vertebral column. The intercostal muscles are divided posteriorly after being separated from the associated intercostal vessels and nerves which are carefully preserved.

The subcostal space is irrigated, hemostasis achieved as carefully as possible, and an Ivalon sponge is tailored to fit. Extra sponge is added anteriorly or laterally between the original sponge and the rib cage to achieve the desired compression. Penicillin and streptomycin, in relatively large quantities, are injected into the sponge and the sponges are sutured in place to each other and/or to adjacent ribs. A single drainage tube is inserted into the subcostal space through an inferior stab wound in the midaxillary line and attached to a water-seal drainage system. If the pleura has been opened accidentally, a thoracotomy tube is inserted intrapleurally and connected with a drainage bottle. The wounds are closed in layers, and a pressure dressing and a snug-fitting chest binder are applied. Drainage tubes are removed when they cease to drain (usually about the third postoperative day). It is quite routine to

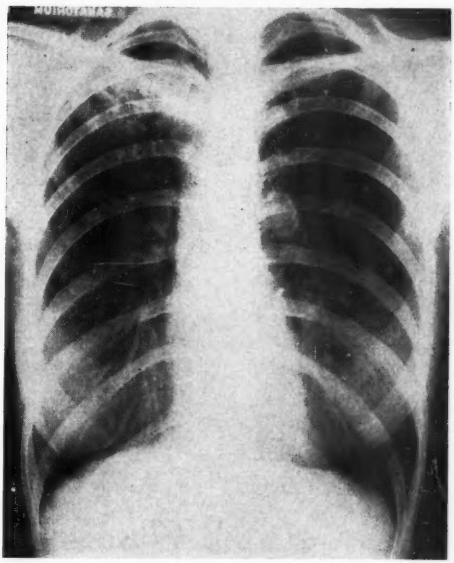

Fig. 2A.— Preoperative roentgenogram of M. J. J., a 21-year-old white woman with residual apical fibrocavitary disease.

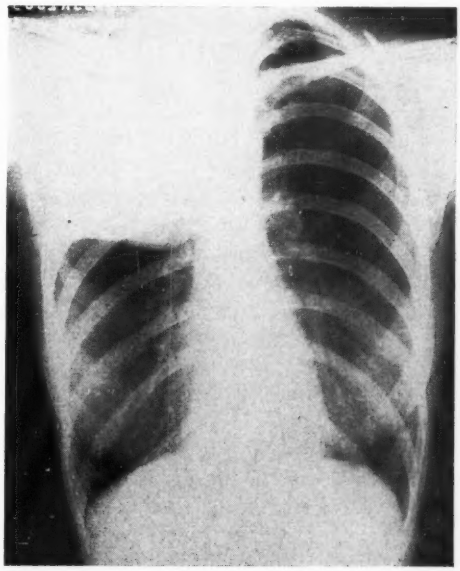

Fig. 2B.—Postoperative roentgenogram after 6-rib plombage thoracoplasty.

collect 800 to 1,000 c.c. of serosanguineous drainage from the wound. If this is not drained, more collapse of the lung is obtained than is desired, and this extra fluid may add to the dangers of infection (Figs. 2 and 3).

The method of introducing the primary extrapleural plombage varies in that a short posterior incision is made and a 10 cm. length of the fourth rib is resected. The endothoracic fascial plane is entered and the parietal pleura freed by blunt dissection over the entire area which is to be collapsed. The opened space is then filled with the shaped sponge which may be sutured to the chest wall. The usual penicillin and streptomycin are introduced into the sponge and the area drained as in the extraperiosteal plombage. Postoperative treatment is the same (Figs. 4*A* and 4*B*).

Insertion of the plombage in the extrapleural space following resection is usually done at the time of resection. Following completion of the lobectomy, the pleura is separated from the chest wall beginning at the site of the thoracotomy and extending over the same area as was filled by the resected lung. A sponge is cut to fit the extrapleural space and sutured in place. After a drainage tube has been inserted anterolaterally, the edge of the pleura is sutured back to the chest wall, extrapleuralizing the entire sponge. In those cases where the pleura has been torn or where a partial pleurectomy was required in the original resection, the pleura is repaired in order to be certain that the sponge is walled off from the remaining lung tissue. Only occasionally has there been insufficient pleura to cover the sponge. In these cases, it is usually preferable to do a plombage thoracoplasty or to insert the extrapleural sponge at a later date (Figs. 5*A* and 5*B*).

Most of the plombes used with resection have been with upper lobectomies. However, three sponges have been used following lower lobectomy. One of these was placed in the apex above a depressed right upper lobe after bilobectomy, and two were placed in the extrapleural space formed by dissecting the lower posterolateral pleura free as far as the edge of the diaphragm and then inserting the sponge in this pocket. The sponge extended over the posterior half of the diaphragm, thus leaving the diaphragm intact and functioning over its anterior half. No ill effects have been noted.

In cases where it is desirable to achieve simultaneous collapse with a resection, but most of the parietal pleura has been stripped off or there has been contamination of the pleural space, a plombage thoracoplasty is performed. This secondary thoracoplasty is carried out in a manner similar to the primary plombage thoracoplasty, except that an attempt is made here to follow the limits of the removed lung tissue rather than square off the lower margin as is done in primary thoracoplasties.

Postresection plombages as second procedures have been carried out for the same indications as those above except in situations where the filling ability of the lung was misjudged, where the original procedure had been of considerable duration, or where there was considerable contamination. As a general rule, this is an unsatisfactory procedure since it is difficult to follow the outlines of the resected lung tissue at a second operation and it probably collapses considerable usable lung tissue.

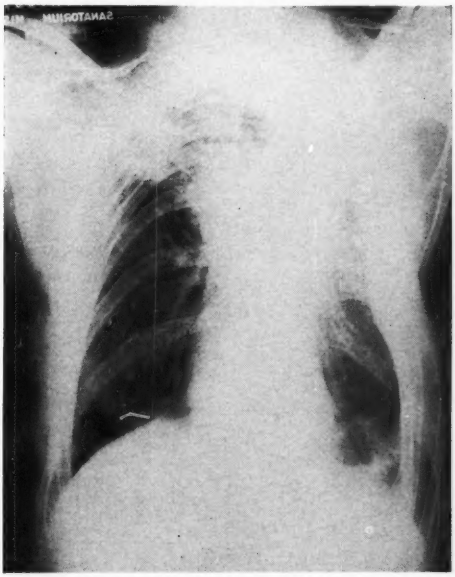

Fig. 3A.—Preoperative roentgenograms of C. W., a 44-year-old white man, who had had a left three-stage 8-rib thoracoplasty five years previously. Patient has a marked kyphoscoliosis and a maximum breathing capacity reduced to 60 L. per minute.

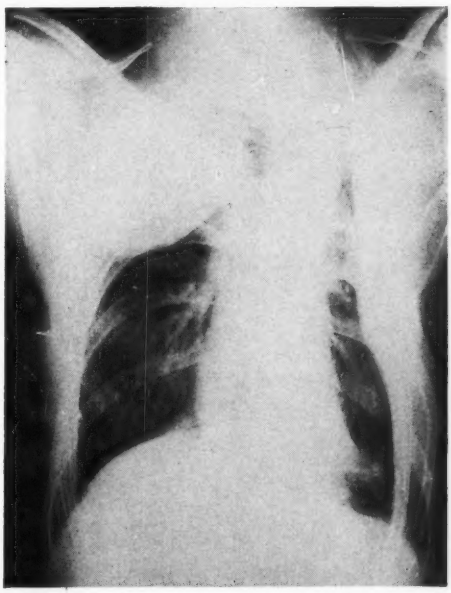

Fig. 3B.—Roentgenogram after right 7-rib plombage thoracoplasty was performed for the fibrocavitary residual with conversion of the sputum and no change in the maximum breathing capacity.

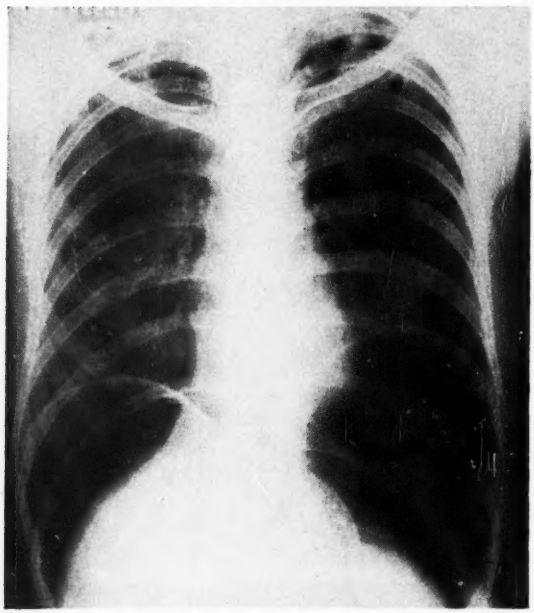

Fig. 4*A*.—Preoperative roentgenogram of C. G., a 40-year-old white man with bilateral apical fibrocavitary disease.

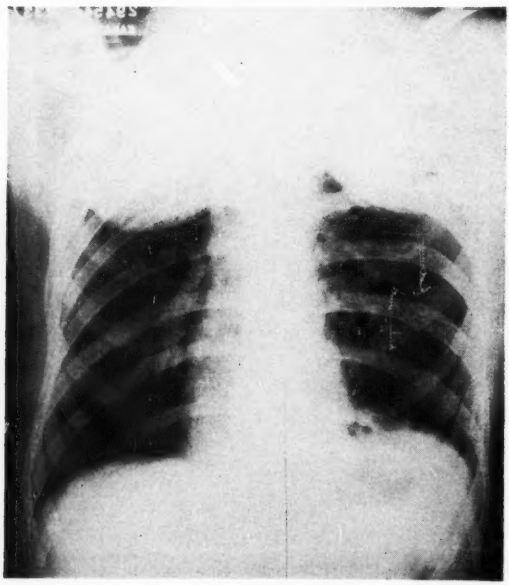

Fig 4*B*.—Roentgenogram after right extrapleural plombage and left plombage thoracoplasty. The sputum converted and there is no clinical or laboratory evidence of respiratory embarrassment.

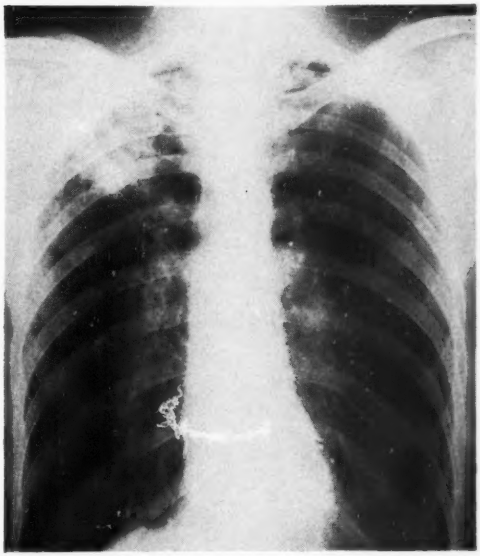

Fig. 5A.—Preoperative roentgenogram of J. W. A., a 54-year-old white man, with an undiagnosed lesion in the right upper lung.

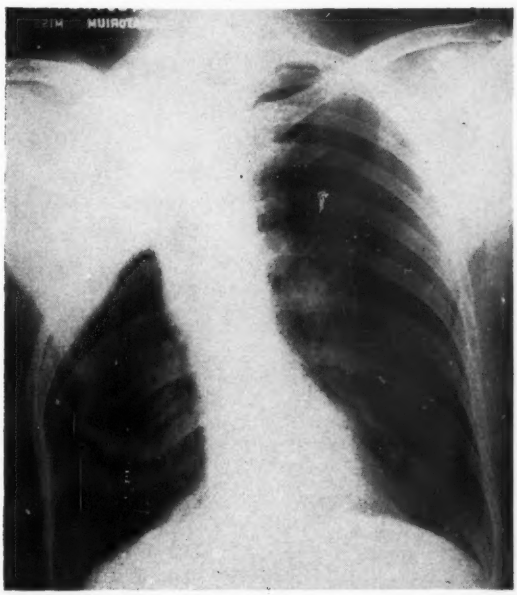

Fig. 5B.—Postoperative roentgenogram after resection of the right upper and right middle lobes for a tuberculous granuloma with a concomitant 5-rib plombage thoracoplasty. The patient continues to do well three years later.

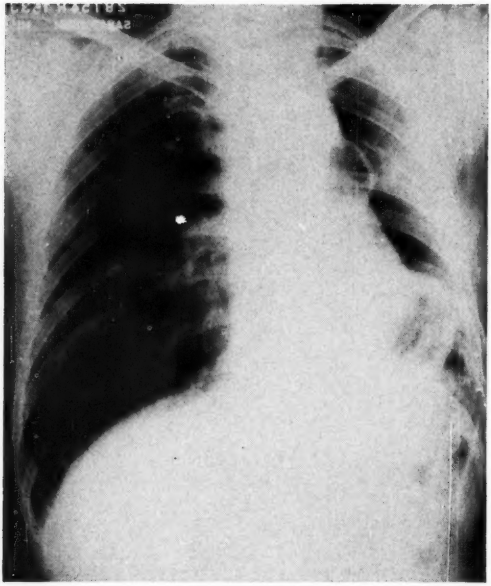

Fig. 6A.—Roentgenogram of W. K. C., a 58-year-old white man, following left pneumonectomy for destroyed lung with sputum positive with resistant bacilli. Note mediastinal shift and marked herniation of lung through anterior mediastinum. Patient had right heart failure in early postoperative period.

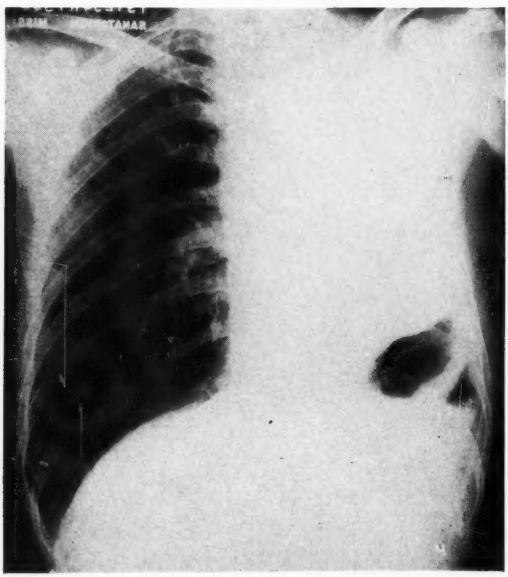

Fig. 6B.—Roentgenogram after insertion of Ivalon sponge into left pleural space three months postpneumonectomy. Patient became much less dyspneic, though symptoms of cor pulmonale have persisted intermittently during the subsequent two years. Sputum remains negative.

The last method of plombage concerns its intrapleural use following pneumonectomies. Most of these pneumonectomies have been for destroyed lungs; and the parietal pleura, or a large portion thereof, has been removed with the diseased lung. Since it is felt that in many cases the field may be contaminated, no sponge has been inserted intrapleurally at the time of the primary operation but is inserted at a second procedure at least three months later. As the majority of bronchopleural fistulas or empyemas will become evident within this time, it seems safer to wait this period. Gale and associates[21] have warned about the increased dangers of infection if a sponge is placed intrapleurally at the time of operation.

Since the sixth rib has usually been resected for the pneumonectomy, the chest is re-entered through an interspace, as resection of the fifth rib creates sufficient deformity for the scapula to impinge beneath the seventh rib. At the time of the second operation, the coagulated fluid is removed and examination of the space demonstrates a thick fibrous peel over the entire chest cavity, almost invariably drawing the mediastinum toward the operated side for a distance of 2 to 5 cm. The fibrous peel is separated at its mediastinal edges, thus allowing the mediastinum to return to the midline. A sponge is then cut to fit the space snugly (but not tightly); penicillin and streptomycin are added, and the chest wall is closed in layers with drainage (Figs. 6*A* and 6*B*).

RESULTS

One hundred and fifty plombage procedures had been performed from one to four years prior to the completion of this study. Seventy-five have been primary plombage thoracoplasties and 10 have been primary extrapleural plombages. There have been 31 extrapleural plombages performed as a single procedure with a resection, and 12 plombage thoracoplasties simultaneous with resection. Of these latter, 4 were associated with pneumonectomies while the others were with lobectomies or their equivalent. There has been only one postresection extrapleural plombage performed as a second operation. Of 9 postresection plombage thoracoplasties, done as second-stage operations, 5 followed pneumonectomies, and 4 followed lobectomies. Twelve intrapleural plombages have been used, all following pneumonectomies.

TABLE I. IVALON SPONGES USED AT M.S.S.
July 1, 1952—June 30, 1955

Plombage thoracoplasty, primary	75
Extrapleural plombage, primary	10
Concomitant lobectomy and extrapleural plombage	31
Concomitant lobectomy and plombage thoracoplasty	8
Concomitant pneumonectomy and plombage thoracoplasty	4
Postlobectomy extrapleural plombage	1
Postlobectomy plombage thoracoplasty	4
Postpneumonectomy plombage thoracoplasty	5
Postpneumonectomy intrapleural plombage	12
Total	150

Infection has been the chief concern. However, to date, only 3 sponges have had to be removed for infection. Two had been used for plombage thoracoplasties and started draining five and twenty-four months, respectively,

postoperatively. The sponges were easily removed and the ribs resected in a conversion thoracoplasty with early healing. One extrapleural sponge inserted at the time of a left upper lobectomy became infected and the patient developed a bronchopleural fistula three months postoperatively. The sponge was removed and the space drained, with spontaneous closure of the bronchopleural fistula the following day. A limited thoracoplasty subsequently has given complete healing. In no case has there been a tuberculous infection.

We have been willing to accept a small percentage of infections, feeling that the advantages outweigh this disadvantage. The chest becomes stabilized in two or three months, and it is a simple matter to remove the sponge and convert to a conventional thoracoplasty should infection intervene.

Desforges, Gibbons, and Strieder[25] have reported finding positive cultures for acid-fast bacilli in the subcostal fluid in 12 of 32 cases of plombage thoracoplasty, done essentially by the same technique as just described, but using Lucite balls. As the tubercle bacilli must certainly be present in the region of the sponge in many of our cases, it has been surprising that none have developed a clinical tuberculous infection. Perhaps the fact that the Ivalon sponge is incorporated into the body tissues, and does not create a permanent nidus of fluid, may in part account for this.

There has been one death associated with these procedures. In this case, cardiac arrest occurred just as the skin had been closed following insertion of a sponge intrapleurally three months after a right pneumonectomy. The incision was immediately reopened, but some extra time was required in removing the sponge before cardiac massage could be started. A full physical recovery was obtained, but there was marked cerebral damage. At the end of one month, during which gradual mental improvement had been noted, sudden death occurred due to a pulmonary embolus, proved by autopsy.

We have had one other complication, this being in a patient who had a pneumonectomy and subsequent 9-rib plombage thoracoplasty. Six weeks following the thoracoplasty, the patient began to complain of numbness, tingling and, later, weakness in the ulnar side of her arm, leading to her later hospitalization at an outside hospital. Subsequently, operation demonstrated a dense layer of fibrous tissue surrounding the third trunk of the brachial plexus which was freed with gradual subsidence of symptoms.[26]

Of some concern to us at the beginning of this series was the fate of the ribs stripped of their periosteum and, indeed, the first thoracoplasties were carried out with complete stripping of the ribs. Many of these ribs atrophied to the extent that they broke spontaneously. However, ribs that have been stripped of periosteum only from the under surface do not atrophy and appear quite normal both histologically (Fig. 7) and roentgenographically.

Some of the sponges have been re-examined as long as twenty-six months following their insertion. If infected, the sponge lay in a sea of purulent fluid and could be easily lifted out. Usually, however, the sponges were firmly fixed by an ingrowth of fibrous tissue for 5 to 10 mm., appeared grossly quite

normal and retained the original resiliency. They were moist, but not accompanied by overt fluid accumulations. Some mild pleuritic inflammation appeared to have been incited, as there was always a moderately adherent symphysis of the visceral and parietal pleurae underlying an extrapleural sponge.

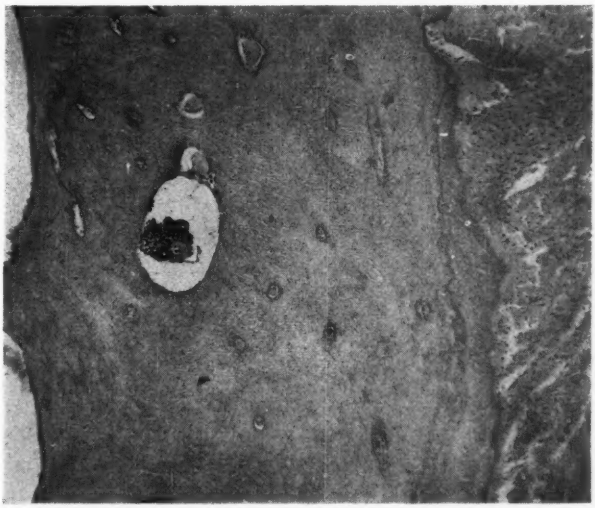

Fig. 7.—Photomicrograph (×200) of rib that had been completely deperiostealized twenty-six months previously at postpneumonectomy plombage thoracoplasty (L. B. B.). The bone appears viable with normal marrow elements. There is dense fibrous tissue surrounding the rib instead of periosteum. Grossly, this rib had atrophied to about one fourth of its previous diameter.

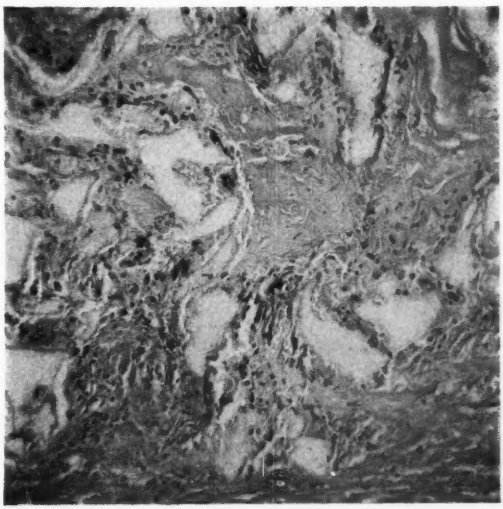

Fig. 8.—Photomicrograph (×200) of Ivalon sponge (light areas) enmeshed with fibrous connective tissue of moderate vascularity. Foreign body giant cells are present.

Microscopically, the ingrowth of fibrous connective tissue was demonstrated with varying numbers of foreign body giant cells and collections of hemosiderin pigment (Fig. 8). The infected sponges showed a tremendous increase in pigmentation and foreign body reaction.

The therapeutic results of the primary plombage thoracoplasties and primary extrapleural plombages as definitive procedures for tuberculosis have been gratifying. Of the 80 patients with 85 primary plombage procedures followed from one to four years, 72 have had their disease controlled by the collapse alone. One continues to have a positive sputum intermittently, but is deemed unsuitable for any further procedure. The other 7 have required additional surgery and a full account of this experience will be the subject of a subsequent report.

Recently it has been demonstrated that virtually any plastic polymer implanted into the subcutaneous tissues of rats will act as a carcinogenic agent, usually producing a fibrosarcoma.[27, 28] These various ploymers included Cellophane, Dacron, polyethylene, polyvinylchloride, Silastic, Pliofilm, nylon, polymethyl methacrylate, polystyrene, Saran, Ivalon, Kel-F, Teflon, and even silk. These agents required roughly fourteen to twenty-eight months to produce tumors, thus usually taking over one-half the life span in a susceptible strain of rat. Whether this can be applied to human experience cannot be readily determined, but it would seem wise, perhaps, to restrict the use of any implanted plastic material in clinical use to the older age groups.

SUMMARY AND CONCLUSION

The history of the development of the thoracoplasty and other space-filling devices in the treatment of tuberculosis is briefly reviewed. Experience with the use of 150 Ivalon (formalinized polyvinyl alcohol) sponges, used as space-taking devices in 144 patients over a period of three years at the Mississippi State Sanatorium, is reported. Of these 150 sponges, 75 were primary plombage thoracoplasties, 10 primary extrapleural plombages, and the remainder postresection plombages used either concomitantly with the resection or as a second procedure.

Of these sponges, 3 have become infected. Following their removal and conversion thoracoplasty, healing has occurred in all. No tuberculous infection has been encountered. One other sponge was removed because of fibrosis involving the third trunk of the brachial plexus.

Plombage has proved itself a desirable operation in the treatment of pulmonary tuberculosis, both for primary collapse therapy and as a space-filling device following resections. While the final evaluation of a foreign substance cannot be determined by four years' observation, the Ivalon sponge appears to be a satisfactory material with an acceptably low incidence of complications.

ADDENDUM

There have been no further complications due to the implanted sponges to date. (Feb. 20, 1957.)

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PLOMBAGE IN THE SURGICAL TREATMENT OF PULMONARY TUBERCULOSIS

A Study of 400 Cases

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THE purpose of this work is to show the advantages and disadvantages of the use of methylmethacrylate (Lucite) balls in the surgical treatment of pulmonary tuberculosis, with special consideration of the long-term tolerance to such a prosthesis. This will be an opportunity to state our position as to the present indications for surgical collapse therapy vs. lung resection.

We present the results of the first 400 operations in which one of us (H. J.) used a prosthesis. There were 345 lung collapses and 55 resections performed between January, 1950, and May, 1955. We excluded from the study the cases in which the balls were deliberately removed within a few weeks after the operation, for example, when they were inserted during a first-stage thoracoplasty and removed at the second stage.

We used hollow spheres, 2.8 cm. in diameter, made of pure methylmethacrylate without the addition of any other plastic material. They can be sterilized in boiling water without alteration in shape.

A spongy material, Ivalon, did not prove satisfactory. The sponges absorbed bloody fluid, their weight increased up to ten times, and fever and pain were observed during the postoperative course.

A material molded and coated with Polystan was discarded because of its heavy weight and its stiffness, which made it hard to position and dangerous to neighboring structures.

SURGICAL COLLAPSE WITH PLOMBAGE

Our 345 cases of collapse with permanent prosthesis include 107 one-stage thoracoplasties, 75 multistaged thoracoplasties, 157 extramusculoperiosteal pneumolyses ("bird-cage" of Lucas and Cleland[6]), and 6 cases of revisional surgery. Table I compares these different types of collapse therapy with permanent plombage over a period of six years.

1. *Technical Details.*—In one-stage thoracoplasties, some ribs are resected, others are simply ruginated. One tries to obtain a satisfactory collapse with as economical a

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TABLE I. DISTRIBUTION PER YEAR OF THORACOPLASTIES AND EXTRAMUSCULOPERIOSTEAL PNEUMOLYSES, WITH AND WITHOUT PERMANENT PLOMBAGE

OPERATIONS	YEARS					
	1950	1951	1952	1953	1954	1955
One-stage thoracoplasties with permanent plombage	1	13	37	32	25	6
Multistage thoracoplasties with permanent plombage	7	15	41	11	–	–
Extramusculoperiosteal pneumolyses with plombage	3	30	23	29	48	48
Revisional collapses with permanent plombage	3	3	–	–	–	–
One-stage thoracoplasties without permanent plombage	9	8	7	5	4	11
Multistage thoracoplasties without permanent plombage	67	29	15	1	1	1
Total operations: 533 Totals	90	98	123	78	78	66

rib resection as possible. In most of our cases, the posterior arc of the fourth rib, the posterior and lateral aspects of the third rib, and the greater part of the second rib were resected, plus a costovertebral disarticulation. Then the lateral border and the inferior face of the first rib were ruginated. In some cases, the posterior arc of the fourth rib was ruginated but not resected. The intercostal muscles were cut at the level of the lateral border of the paravertebral muscles but were not severed from the lateral aspect of the pleura and their vessels were carefully preserved to maintain a good blood supply to the muscles and to the pleura. One may lower the apex slightly by blunt dissection with a small sponge on a clamp but should avoid a too extensive liberation of the lung on its mediastinal aspect. Thus, the space for the prosthesis is limited above by the inferior face of the first rib which separates it from the vessels and nerves of the base of the neck, and medially and below by the intercostal muscles and periosteal beds which protect the mediastinum and the lung. The space is loosely filled with Lucite balls, varying from eight to twelve in number, according to the volume of the space. Such a thoracoplasty collapses the upper third of the lung. We use it for small apical lesions with cavitation.

Multistage thoracoplasties with permanent plombage were used from 1950 to 1953, inclusive, following which extramusculoperiosteal collapse was preferred, because thoracoplasties of six ribs or more required several operations, involved the base of the lung, and impaired respiratory function.

Extramusculoperiosteal pneumolyses have always been followed by plombage. Ribs were neither cut nor removed, but ruginated, usually five to seven of them, the liberation being pushed far forward at the level of the upper ribs. Care was taken, as in thoracoplasties with plombage, to preserve a continuous wall of intercostal muscles and periosteal beds protecting the mediastinum and the lung. The lateral wall of the space is rigid, being composed of the bare ribs. The volume of the plombage should be such that it would collapse the diseased lung completely and prevent any re-expansion of the lung along the mediastinum. It should exert no pressure upon the mediastinal vessels. The usual number of balls inserted is from twenty to thirty. Such an operation collapses at least one half of the lung and does not interfere with the function of the base. It is used in the treatment of cavitary lesions requiring an extensive collapse.

Revisional collapses were few. All were done, in 1950 and 1951, for cavities persisting after a previous thoracoplasty. The cavities were of small size, situated some distance from the pleura, and presented no indication for resection. In these cases, extrapleural pneumolyses with permanent plombage were used.

2. *Complications.*—There were three early deaths (0.9 per cent). All of them occurred in patients who had had multistage thoracoplasties performed and were over 40 years of age. The operations were attempted despite the presence of cardiorespiratory impairment proved by electrocardiography or respiratory function studies.

Aggravation of pulmonary tuberculosis occurred in 8 patients (2.3 per cent). It was seen in cases of extensive collapse by multistage thoracoplasties or extramusculoperiosteal pneumolyses. Finally, there were two satisfactory end-results, two unsatisfactory end-results, and four deaths.

Infection of the space occurred in 12 patients (3.4 per cent). It was a staphylococcic infection, observed nearly always in the years 1950 to 1952 and complicating, equally, the different types of collapses; possibly too much confidence had been placed in prophylaxis of infection by the use of antibiotics. Later on, with extreme aseptic care and isolation of patients during the postoperative period, infections became very rare (0.6 per cent). The ultimate results in cases complicated with infection were related to the severity of the pulmonary tuberculosis and to the efficacy of the collapse therapy; 7 patients were cured by antibiotics alone or combined with the removal of the plombage. One patient had an unsatisfactory result. Four patients died later. We did not see any early tuberculous fluid or sterile empyema.

There were 3 cases of phlebitis (0.9 per cent), apparently unrelated to the use of a plombage.

Migration of balls was a severe complication only once. The parietal pleura had been damaged during the operation and had been sutured. The suture gave way later and the migration of balls into the pleural cavity required a thoracotomy. In subsequent operations, when the pleura had been damaged, the balls were placed in a nylon net. We preferred this solution to collapse therapy without plombage because the balls help to correct the pneumothorax resulting from the pleural tear and to keep bloody fluid from entering the pleural cavity.

The migration of balls through the intercostal spaces was observed in a few patients. After an extramusculoperiosteal pneumolysis, one or two spheres may go through the anterior interspaces. If this is noticed early enough, pressure with the hand makes the ball go back into the space. Soon the thoracic wall becomes rigid and no migration is possible.

From three to six months after a thoracoplasty, balls may escape into the axilla or beneath the scapulo-vertebral muscles. Rarely will more than one or two spheres escape; the ones that did were removed under local anesthesia. In 2 cases, a lung which could not be collapsed because of a rigid pleura was responsible for the migration of the whole plombage, which had to be removed.

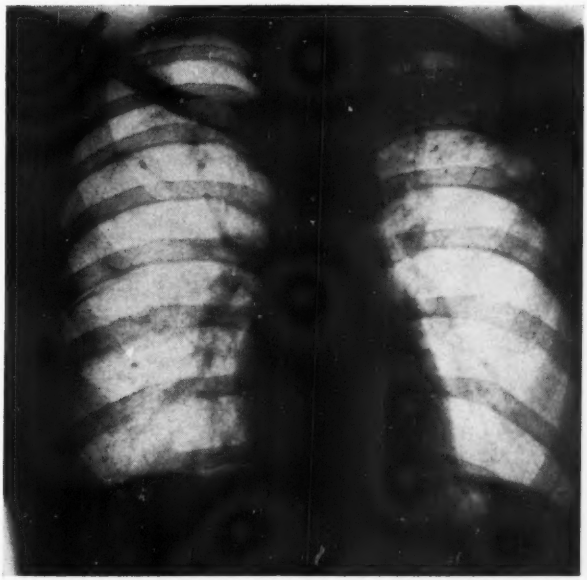

Fig. 1.—X-ray of a 24-year-old woman with bilateral pulmonary tuberculosis of 7 years' duration. Right artificial pneumothorax. Cavity in left apex. Low respiratory reserves.

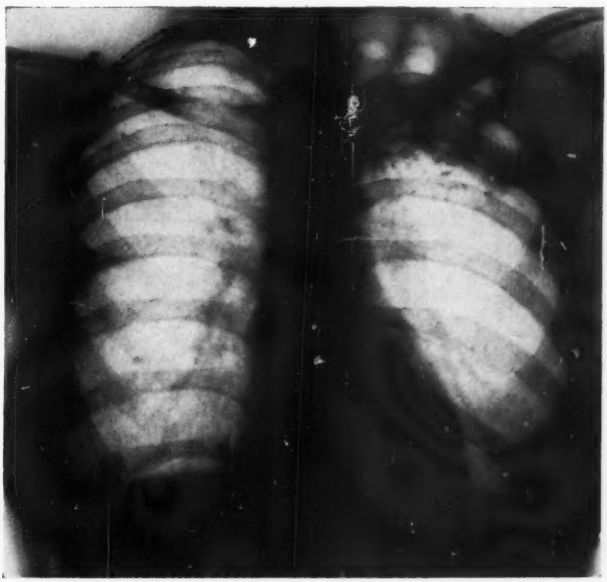

Fig. 2.—X-ray of the same patient (Fig. 1) after left upper thoracoplasty was done in June, 1953. The second and third ribs were resected, the first and fourth ribs were ruginated, and 12 Lucite balls were inserted. Arrested case.

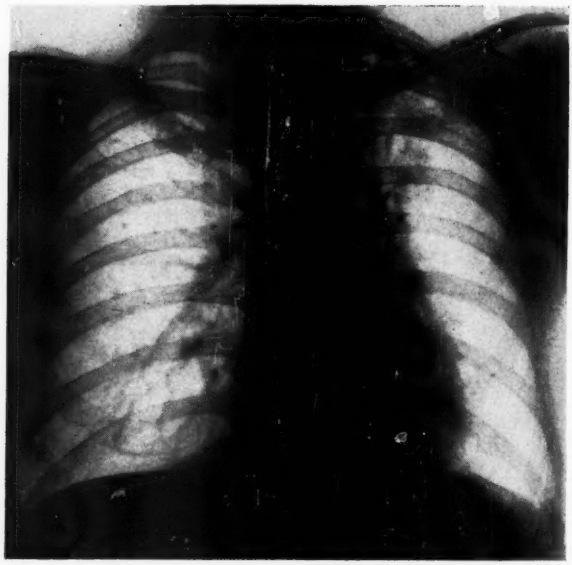

Fig. 3.—X-ray of a 46-year-old man who had left pleurisy and tuberculosis of the knee in 1926, left pulmonary tuberculosis in 1933, and relapse in the left apex with cavitation in 1953. Low respiratory reserves (vital capacity—2,100 c.c.).

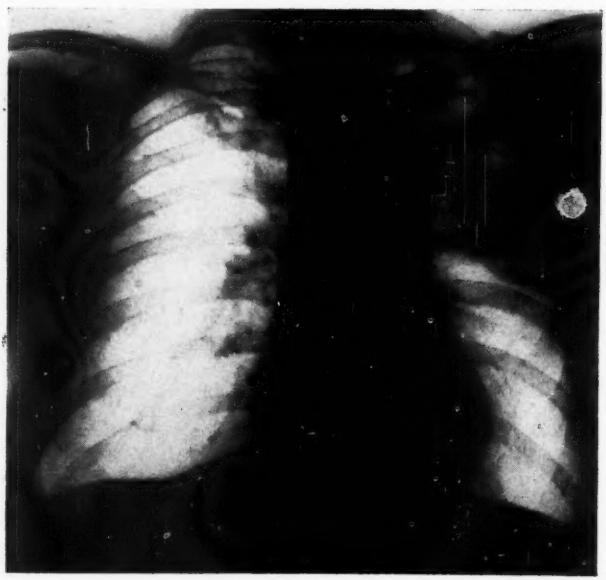

Fig. 4.—X-ray of the same patient (Fig. 3). An extramusculoperiosteal pneumolysis was performed in May, 1954. Five ribs were ruginated, and 25 Lucite balls were inserted. Arrested case. Patient resumed work in March, 1955.

No spheres escaped toward the mediastinum, the neck, or the lung.

Thus, the postoperative complications in 345 cases of collapse with permanent plombage resulted in eleven deaths (3.2 per cent) and three unsatisfactory results (0.9 per cent). These fourteen bad results followed a multistage thoracoplasty in ten instances (13.3 per cent) and an extramusculoperiosteal pneumolysis in four instances (2.5 per cent). Twelve of them were observed after operations done during the period 1950 to 1952 (6.8 per cent) and twice after operations performed during the period of January, 1953, to May, 1955, (1.2 per cent).

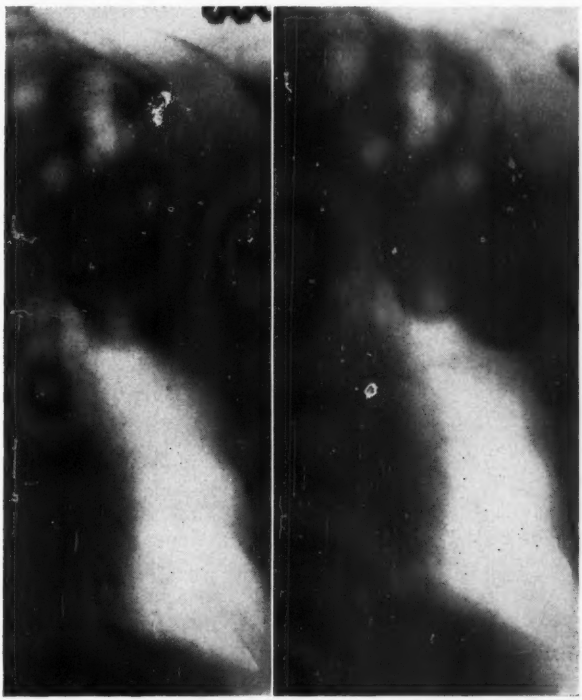

Fig. 5.—X-ray of the same patient (Fig. 3). Tomograms taken in May, 1956, two years after the operation. The collapse under the prosthesis is excellent, the formerly diseased lung has not re-expanded.

3. *Late Results.*—The evaluation of late results, made in 1956, gave a follow-up of one year minimum to six years maximum.

Of a total of 345 patients operated upon, we obtained information on 301, or 88 per cent. For the other 44 patients, the information obtained was not sufficient. When leaving the sanatorium or when last heard of, there were 29 arrested cases, 13 fairly satisfactory results, and 2 unsatisfactory results. The follow-up period was not considered to be long enough for these cases to be included in Table II.

TABLE II. PRESENT CONDITION OF 301 PATIENTS WITH LUNG COLLAPSE AND PERMANENT PLOMBAGE

OPERATIONS	NUMBER OF PATIENTS	ARRESTED CASES			IMPROVED CASES	UNSATISFACTORY RESULTS	DEATHS
		AFTER COLLAPSE	AFTER RESECTION	TOTAL AND PER CENT			
One-stage thoracoplasties	87	68	7	75 (86%)	12 (14%)	0	0
Multistage thoracoplasties	66	39	4	43 (65%)	10 (15%	4 (6%)	9 (14%)
Extramusculoperiosteal pneumolyses	144	104	8	112 (78%)	21 (15%)	6 (4%)	5 (3%)
Revisional collapses	4	4	0	4	0	0	0
Totals	301	215	19	234 (79%)	43 (14%)	10 (3%)	14 (4%)

We listed as arrested cases those in which there were negative bacteriologic tests and no evidence of activity, demonstrated by repeated x-rays. Most of these ex-patients had resumed an active life. It should be noted that in 19 cases the tuberculosis was arrested only after a resection had been performed subsequent to the lung collapse.

We listed as improved cases those patients in whom a definite improvement was seen following surgical collapse with plombage, but who had an occasional positive sputum or in whose x-rays questionable shadows were seen. In the cases in which resection was contraindicated, the patients certainly benefited from the collapse therapy. In some cases, the positive bacteriologic tests were explained by contralateral lesions.

Results were considered unsatisfactory in cases where there was no definite improvement from the collapse or where there was an early or late relapse.

Deaths include those of the postoperative period and later ones. We excluded from the study four deaths from accidents in ex-patients in whom the disease had been completely arrested and who had resumed an active life. Table III gives a detailed study of the deaths in our series as related to the type of collapse therapy, date of operation, and time of death in relation to the operation.

TABLE III. DEATHS AS RELATED TO YEAR OF OPERATION AND TYPE OF COLLAPSE (PREOPERATIVE USE OF INH BECAME COMMON FROM JANUARY, 1953, ON)

YEARS	TYPE OF COLLAPSE	NUMBER OF PATIENTS	EARLY DEATHS	LATER DEATHS	TOTAL AND PER CENT OF DEATHS	
Jan., 1950, to Dec., 1952	One-stage thoracoplasty	51			0	12 (6.8%)
	Multistage thoracoplasty	63	3	5	8 (12.7%)	
	Extramusculoperiosteal pneumolysis	56		4	4 (7.1%)	
	Revisional collapse	6			0	
Later	One-stage thoracoplasty	65			0	2 (1.2%)
	Multistage thoracoplasty	12		1		
	Extramusculoperiosteal pneumolysis	101		1	1 (8.5%) 1 (1%)	
	Totals	354	3	11	14 (4%)	

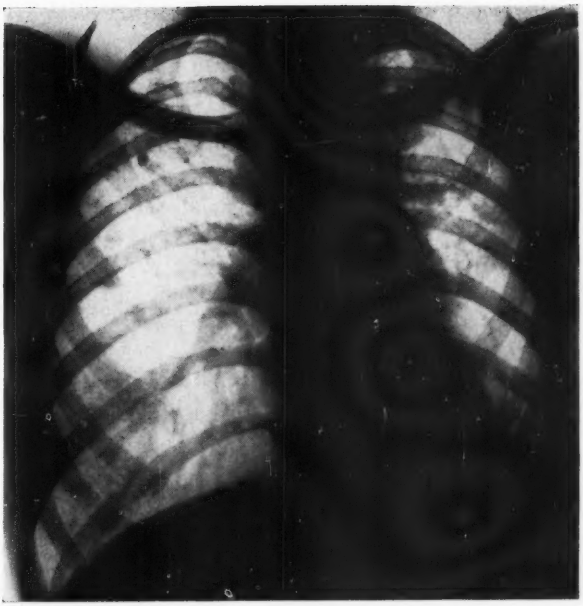

Fig. 6.—X-ray of a 35-year-old woman which shows extensive lesions of the left lung with cavitation and spread to the right lung. Undisciplined patient, unable to take strict bed rest.

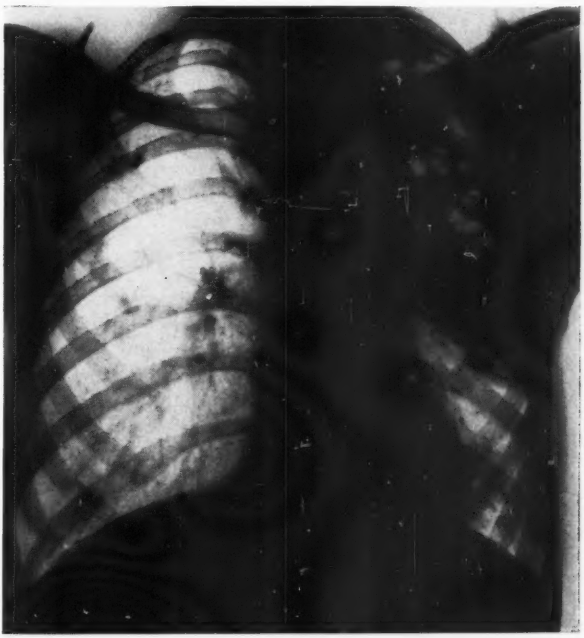

Fig. 7.—X-ray of the same patient (Fig. 6) after an extramusculoperiosteal pneumolysis performed in January, 1953. Seven ribs were ruginated and 33 Lucite balls were inserted. Improved at the present time.

The important differences in results, according to type of operation as shown in Tables II and III, are explained by differences in the severity of the disease. One-stage thoracoplasties were used in the treatment of apical lesions of moderate severity, already improved by medical treatment, and often surgery was performed only as protection against a possible relapse.

Multistage thoracoplasties on the other hand were used in more extensive and more severe cases, and in addition most of them were performed before isonicotinic acid hydrazide (INH) was available.

Extramusculoperiosteal pneumolyses were done in similar cases but only after some improvement was obtained from antibiotic therapy. This type of collapse is not very traumatic.

Fourteen patients were treated with bilateral collapse therapy. Eight of them had a thoracoplasty on both sides; 4 had an extramusculoperiosteal pneumolysis on both sides; 2 had a thoracoplasty on one side and an extramusculoloperiosteal pneumolysis on the other. As far as indications were concerned, 5 of the patients were more than 40 years old, and 7 had additional trouble such as respiratory insufficiency, alcoholism, among others. The overall results were good; 8 arrested cases, 5 improved cases, 1 unsatisfactory result.

4. *Tolerance to Permanent Plombage.*—Our study reveals the extremely good tolerance of the organism to Lucite balls.

A late infection of the plombage space was observed in 2 patients only. One of them had had a one-stage thoracoplasty and the disease was completely arrested. A year later a pneumococcal infection of the space, secondary to a general infection, was observed. It healed under antibiotic treatment and after removal of the plombage. The other late infection was seen in a man who had had a bilateral operation for severe cavitary disease with an unsatisfactory result. A late bronchial fistula appeared under an extramusculoperiosteal pneumolysis, requiring the removal of the plombage and drainage. The prognosis in this case is poor.

Aseptic osteolysis of ruginated ribs was seen in several patients who had had extramusculoperiosteal pneumolyses performed without any apparent adverse effect.

We do not see any late migration of spheres toward the mediastinum, lung, or neck.

Attention was paid to pain related to a permanent plombage. Fifty-two per cent of the patients experienced no pain; 37 per cent mentioned slight and intermittent pain, mostly related to a change in weather and excessive fatigue; and 11 per cent complained of definite pain with radiation along the ulnar nerve. So far we have had to remove the plombage, because of pain, in only one patient.

PLOMBAGE ASSOCIATED WITH PULMONARY RESECTION

In 55 patients in whom pulmonary resection had been performed, Lucite balls were placed in the hemithorax after the operation to help fill

the residual space. The prosthesis was inserted either immediately within the pleural cavity or, in a later stage, in the thoracic wall.

1. *Intrapleural Plombage Following Resection.*—In 18 patients who had had pneumonectomies performed, balls were placed at the end of the operation. Two of these died, one soon after the operation, the other one some time after; it does not seem that the use of plombage was, in any way, responsible for these deaths. Three other patients had a small bronchial fistula which led to the removal of the plombage and a total thoracoplasty; it may be that the plombage contributed to some extent to the production of these small late fistulas.

In 18 patients who had had a more or less extensive resection of the upper parts of a lung performed, a plombage was placed both to take the place of the resected lung and to prevent distention of the remaining lung. The spheres were contained in a nylon net which was fastened to the upper intercostal muscles. In 6 of these patients, bronchopleural fistulas occurred, some of them followed by infection of the space. This technique was subsequently abandoned.

2. *Extramusculoperiosteal Pneumolysis With Plombage After Resection.*—In 21 cases of partial or total resection, an extramusculoperiosteal pneumolysis was performed several weeks or months after the operation. Results were very satisfactory. This type of collapse seems to us to be preferable to a thoracoplasty which usually interferes more with the morphology and the function of the thorax.

This was done after pneumonectomies when the fluid in the residual space failed to organize, or as treatment for a late bronchial fistula.

After partial resections, this was done to obliterate a residual air space in the apex, or to check overdistention of the remaining lung after an extensive resection, or to fight a recurrence of pulmonary tuberculosis.

DISCUSSION

We consider the tolerance of Lucite balls by the organism to be demonstrated by our data, provided they are used only in well-defined circumstances. They should not be used in a pocket of extrapleural pneumolysis, when the pleura is thin, and particularly when caseous lesions are present in the lung. They should not be placed directly in contact with the mediastinum, since severe accidents resulting from their migration have been reported. They should not be in contact with the lung, as was shown by our own experience of plombage after partial resection.

Thus, the plombage is to be separated from the lung and mediastinum, in a space in which the medial wall is made of the intercostal muscles and the periosteum. The quality of the operative technique is responsible for the good tolerance of the plombage. Strict asepsis is compulsory to avoid infection from ordinary contaminants. All possible improvement from medical treatment should have been obtained prior to the operation.

As compared to procedures without it, plombage added to surgical collapse prevents re-expansion and allows the attainment of the necessary amount

of collapse in a one-stage operation. The skeleton is least disturbed, the resection of ribs kept to a minimum, or replaced by simple rugination as in an extramusculoperiosteal pneumolysis, and the dynamics of the thorax suffer little damage.

These advantages are of special interest in certain categories of patients. In emphysematous patients, a collapse can be obtained which would be impossible otherwise. In people with weak muscles, women or adolescents, or subjects with some deformation of the spine, the plombage allows the deformities to be kept at a minimum. In patients with bilateral pulmonary tuberculosis, the functional impairment from collapse therapy is reduced and if necessary a bilateral collapse can be done.

When a plombage is associated with an apical thoracoplasty or with an extramusculoperiosteal pneumolysis, without a mediastinal pneumolysis, the collapse is obtained from the upper and lateral parts toward the lower and medial parts, thus differing from the collapse of thoracoplasties with extrapleural pneumolysis of the Semb, or Björk types, which is strictly vertical.

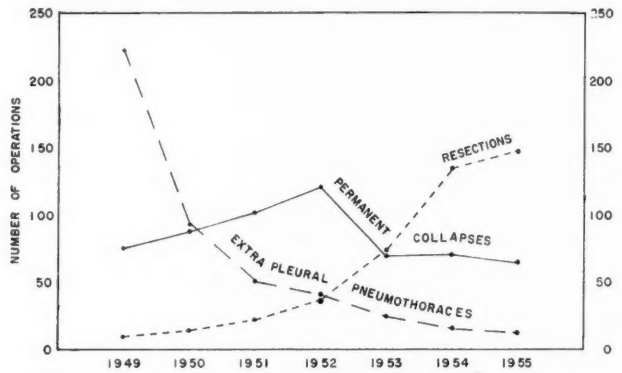

Chart I.—Evolution of different types of operations for pulmonary tuberculosis, 1949 to 1955.

We are far from considering it a disadvantage. When cavities persist after reliable medical treatment, and when the lesions have little tendency to retract, we consider it desirable to obtain a collapse of the diseased lung against the relatively fixed mediastinum rather than against the lower parts of the lung. The diseased lung under the influence of the plombage becomes atelectatic, and the biologic factors of healing are helped.

What should be, at the present time, the relative indications for surgical collapse and resection? When faced with lesions which have not yielded to rest and antibiotics, we usually prefer resection. Chart I shows the evolution of our indications for the years 1949 to 1955.

But, specific indications should be kept for surgical collapse therapy. While classical thoracoplasties can be blamed for excessive deformation and

functional troubles, lung collapse with plombage does not deserve such criticism, and the indication for permanent collapse should be easier to determine.

Generally speaking, collapse therapy should be preferred whenever opening the pleural cavity, manipulating the lung and reducing the respiratory function in the immediate postoperative period, expose the patient to more than the usual risk. More precisely, when the opposite lung is unable to maintain a normal respiratory function by itself, due to pleural or other causes, and, in the presence of emphysema or pulmonary fibrosis, collapse therapy is to be preferred to resection. We prefer the use of lung collapse in patients over 45 years of age, and who have any associated disease.

In patients with disseminated lesions, we do not like to employ multiple resections which leave wide bare surfaces and are frequently followed by a stormy postoperative course.

We prefer collapse therapy in undisciplined patients, because of the risk of a premature return to activity. This would be particularly dangerous after resection, which should be followed, without exception, by several months of postoperative treatment.

Needless to say, only cavitary lesions can be benefited by collapse therapy, solid caseous foci are not likely to be helped.

In some cases, an extramusculoperiosteal pneumolysis with plombage was successfully used either to replace an extrapleural pneumothorax, when it became evident that the collapse should be made permanent, or to obliterate the residual space after a partial resection, or to make the residual space smaller after a pneumonectomy.

SUMMARY AND CONCLUSIONS

1. A study of 400 operations for pulmonary tuberculosis, making use of methylmethacrylate (Lucite) balls as a plombage, is presented. The plombage was extremely well tolerated over a period up to six years, provided it was separated from the lung and mediastinum by a continuous wall of intercostal muscles, with an intact blood supply, and periosteal beds.

2. The addition of plombage to a surgical collapse provides several advantages. It prevents pulmonary re-expansion and reduces to a minimum the morphologic and functional side effects of collapse therapy. At the present time, we do limited thoracoplasties without resection of the first rib, and extramusculoperiosteal pneumolyses without cutting or resecting any rib.

3. It is generally agreed that resection is the best surgical treatment for pulmonary tuberculosis, whenever indicated and possible. However, when surgery is indicated and resection is impossible, or potentially dangerous, thoracoplasty and extramusculoperiosteal pneumolysis with Lucite balls may offer a better chance to the patient than conventional collapse therapy or continued medical treatment and should be considered.

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STREPTOKINASE AND STREPTODORNASE IN THORACIC SURGERY FOR PULMONARY TUBERCULOSIS

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INTRODUCTION

STREPTOKINASE, an exotoxin of a pathogenic strain of hemolytic streptococcus capable of acting as an enzymatic catalyst to prevent blood clotting and to promote the liquefaction of human fibrin, was described in 1933 by Tillett and Garner.[1] Later, McCarty[2] and, independently, Tillett and associates[3, 4] described streptodornase, an exotoxin produced by a strain of hemolytic streptococcus, capable, by enzymatic action, of breaking down nucleoproteins to smaller acid-soluble nucleotides. This property causes the liquefaction of the thick, viscous exudate found in closed infections.

Since this time, there has been a great deal of interest in these enzymes and they have been used topically, by intramuscular injection, and by injection into blood and purulent collections. A very limited bibliography from the fifty-five articles collected by the authors would include References 5 through 10. Finnerty[9] reports success in the treatment of empyema when SK-SD is used in combination with closed tube drainage. Read and Berry[11] have nothing but praise for these lysing agents when used to treat hemothorax. Sherry and Tillett[10] also report glowingly on the success of SK-SD in the treatment of hemothorax, but do indicate that there may be failure in an occasional case. A few of their cases involved clotted hemothorax postpneumonectomy for tuberculosis. No articles were found dealing with the use of the enzymes during the immediate postoperative period in thoracic surgery for pulmonary tuberculosis.

It was the opinion of the staff at this hospital that the results of enzymatic débridement in postoperative hemothorax and hemopneumothorax were, at best, equivocal. In view of the absence of information on the subject in the literature and to check the veracity of our clinical impression, the present study was undertaken.

ANALYSIS OF CASES

In the course of 355 thoracotomies, streptokinase-streptodornase was used in 37 cases complicated by hemothorax or hemopneumothorax. The original operative procedures were 22 segmental resections, 8 lobectomies, 6 decortications and one lobectomy plus segmental resection. In 25 cases, the drug was employed for mild to severe hemothorax and in the remaining 12 cases, hemopneumothorax of varying severity was the presenting complication. These

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complications appeared within the second postoperative day with the exception of one hemothorax following decortication which did not appear until the third day.

The initial use of the drug varied from the first to the seventh postoperative day, the second day after surgery being employed in a majority of cases. Varidase, a product containing 100,000 units of streptokinase and 25,000 units of streptodornase per vial, was used in each case. Generally two and occasionally four vials of freshly prepared solution were introduced into the hemothorax by needle puncture of the clamped-off intercostal drainage tubes, the dosage being divided between the two tubes. A total of 60 c.c. of diluent was employed. An attempt was made to keep the tubes clamped off for at least four hours and aspirin was used as an antipyretic. On occasion, it was necessary to open the tubes after only two or three hours due to fever and chest pain. The systemic effects promptly disappeared after opening the tubes.

In the successful case, unclamping the tubes resulted in a prompt flow of unclotted bloody fluid and air. This would generally subside in twenty-four to forty-six hours and the amount of bloody fluid evacuated varied up to 1,000 c.c. The re-use of the drug depended on the clinical course, result obtained, and appearance of the x-ray. The drug was not used more than four times in any case.

Cases were classified as good if the clot was evacuated, the lung or lung remnant expanded, and no further operative procedures were required. Poor results failed to experience clot removal, and required such operative procedures as decortication or thoracoplasty. Equivocal results were those falling between the above two groups in which the value or lack of value of the treatment was difficult to assess.

Results were classified as good in 24 cases, equivocal in 3 cases, and poor in 10 cases. The presenting complication was hemopneumothorax in 11 of the 13 cases falling in the equivocal or poor category. A temporary increase in the amount of air leakage was the rule after use of the enzymes, but this was self-limiting. In no case did active bleeding recur as a result of the enzymatic action.

TABLE I

COMPLICATIONS		RESULTS AFTER USE OF SK-SD		
		GOOD	EQUIVOCAL	POOR
Hemothorax	25	21	1	3
Hemopneumothorax	12	1	3	8
Total	37	22	4	11

COMMENT

Streptokinase-streptodornase is used to remove the organized blood clot from the hemithorax, including the raw lung surface resulting from the operation. By this procedure, it is hoped that the lung will be given a second chance to expand and fill the hemithorax. As an aid in obtaining this objective, 12 to 14 inches of water negative pressure is applied to the chest tubes in order to

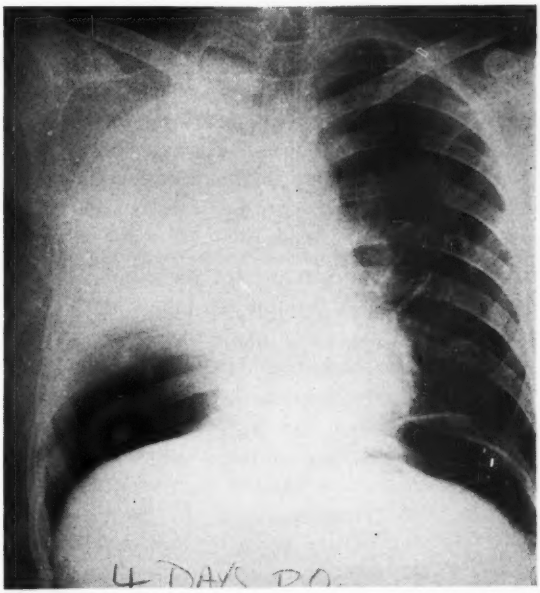

Fig. 1.—Case 16. Hemothorax, right, fourth postoperative day, following right upper lobectomy, thoracoplasty, and pneumoperitoneum.

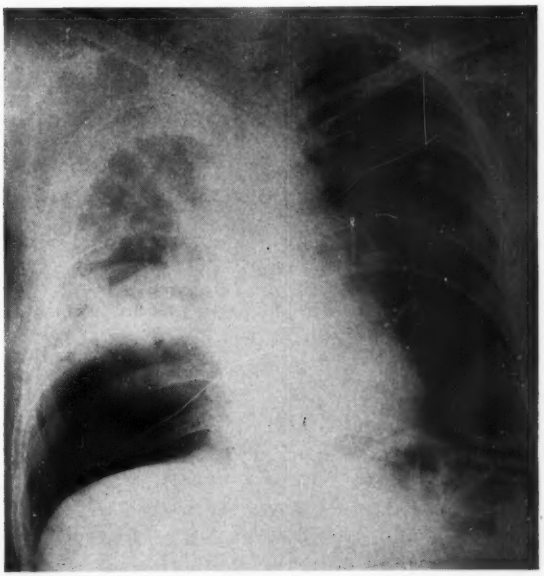

Fig. 2.—Case 16. Result two days later following one application of SK-SD.

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remove the liquefied clot, expand the lung, and remove the air leakage which results from dissolving the clot from the raw lung surface. This leakage of air generally subsides in thirty-six hours.

The best results were obtained in mild to moderate hemothorax when the enzymes were instilled through the chest tubes. Results were poor or equivocal in all but one case of hemopneumothorax. The enzymes did not cause further bleeding in any case and there were no complications of any type resulting from their use.

SUMMARY

1. Streptokinase and streptodornase are useful in treating postoperative hemothorax following resection of tuberculous pulmonary residuals.

2. The enzymes have little value when there exists a large element of pneumothorax in addition to the hemothorax.

3. Best results were obtained when the drugs were used on the first to the third postoperative day.

4. Injection of the drugs through the clamped-off chest tubes is the method of choice.

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LIMITATIONS OF LAMINAGRAPHIC INTERPRETATION IN PULMONARY TUBERCULOSIS

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DECKER and his associates[1] propose the selection of tuberculous cases for excisional surgery of the lung on the basis of the type of lesion remaining after an adequate period of drug therapy. With a large series of patients, and, of necessity, having only radiologic findings to classify the unoperated cases, they apparently demonstrated conclusively that surgery was mandatory in the open-positive and open-negative cases. There was no statistically significant difference in result between the resected and unresected cases on short-term follow-up in those classified as closed-negative.

The reported results were so striking and clear-cut that our selection of cases for excisional surgery at Fitzsimons Army Hospital was definitely influenced. At the present time, tuberculous residuals which are obviously of a caseonodose, or closed-negative nature, are not resected unless they exceed 2 cm. in greatest diameter, that line of demarcation being arbitrarily selected. There is never any disagreement concerning the necessity for surgery in the open-positive case, but the decision for surgery in the open-negative case is often made only after a great deal of discussion and, I am sure, an open-negative case is occasionally not given the benefit of surgery.

To quote Decker and his associates,[1] ". . . open or closed lesions by rigid radiologic examination . . ." gives the impression that radiologic diagnosis is a relatively exact science upon which one can rely. We repeatedly encounter examples of failure of correlation between the interpretation of laminagrams by radiologist, internist, and surgeon, and the findings in the resected specimen. This led to a little self-examination to determine the reliability of chest laminagrams.

Five patients, subjected to excisional surgery only after much discussion and difference of opinion, were selected for the test. These cases were accumulated in a period of three weeks on the Tuberculous Thoracic Surgery Section and represent no unusual situation or biased selection of cases. They were all negative when operated upon and had been for at least five months. Three patients had never been found positive. The common denominator in each was the interpretation of the laminagram. The majority came to surgery because some examiners were not sure there was not a cavity, which is hardly precise, exact, or scientific. The cases were selected by the author, preoperative laminagrams obtained, signs of identification masked, and a group of 13 qualified

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and experienced chest physicians and thoracic surgeons were asked to interpret the laminagrams and state an opinion regarding whether a cavity was or was not present in each case. Table I gives the pertinent part of the pathologic diagnoses and the percentage of correct diagnoses on laminagraphic interpretation.

TABLE I. DIAGNOSES RENDERED FROM LAMINAGRAMS IN 5 CASES OF PULMONARY TUBERCULOSIS WITH PATHOLOGIC DIAGNOSES AND PERCENTAGE OF CORRECT DIAGNOSES

CASE NUMBER	PATHOLOGIC DIAGNOSIS	DIAGNOSIS FROM LAMINAGRAMS		PERCENTAGE CORRECT
		NO CAVITY	CAVITY	
1	Cavitary with bronchocavitary junction	4	9	69
2	Cavitary with bronchocavitary junction	7	6	46
3	Cavitary with bronchocavitary junction	4	9	69
4	Cavitary with bronchocavitary junction	6	7	54
5	Caseonodose	10	3	77

The percentage of correct diagnoses is a little better than one could obtain flipping a coin and could logically lead one to doubt the ability of the clinicians to interpret chest laminagrams, so the diagnoses of the radiologists were examined from their previously rendered reports. Interestingly, their percentage of correct diagnoses was zero. The one case diagnosed by the pathologist as caseonodose was thought to show cavitation and in each of the others the statement is made that no cavities can be seen.

COMMENT

This exposé of the limits of accuracy of chest laminagraphy in selected cases is in no way meant to be a blanket denunciation of the procedure. We all realize that it is often a great aid in precise diagnosis and is usually much more revealing then conventional chest films. Without the assistance given by this procedure, many otherwise unavoidable errors would be made.

However, and this is the point of importance, laminagrams are not infallible and we are forced to believe that the differentiation of the closed and open-negative case is not always a simple matter based on the results of bacteriologic cultures and chest x-ray, to include laminagraphic evaluation.

It is well known that material of different density is required, as a rule, to make a cavity readily visible. Should that cavity be filled with fatty, necrotic debris, contain no air, and have its bronchocavitary junction sealed for the moment, with inspissated material, no cavity will be definitely visualized on laminagrams. However, this blocked bronchocavitary junction might readily be opened incident to the trauma of excision, leading to the finding of an air-containing cavity by the pathologist.

CONCLUSIONS

The differentiation of closed and open cavities by laminagrams is often not possible. This leads to the necessity for re-evaluating the classification by Decker and his associates[1] of cases into open-positive, open-negative, and closed-negative categories.

I am forced to conclude that any patient who was ever cavitary must be considered to be still so and subjected to excisional surgery unless it can be proved beyond any reasonable doubt that the cavity is inspissated, closed, and has no bronchocavitary junction. To state it another way, when in doubt, the patient's best interest will generally be served by excisional surgery.

SUMMARY

1. The fallibility of laminagrams in revealing the presence of cavities in selected cases of pulmonary tuberculosis is demonstrated.

2. The difficulty of distinguishing the open-negative from the closed-negative case is discussed.

3. Surgery in any case which was once cavitary is advised unless cavity closure can be proved beyond a reasonable doubt.

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INDUCTION OF ANESTHESIA IN PATIENTS UNDERGOING THORACIC SURGERY: EXPERIMENTAL STUDIES IN VOLUNTEERS WITH A SYNTHETIC STEROID, VIADRIL

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IN 1941, H. Selye[8] published a series of papers describing his experiments in animals with certain steroids, such as desoxycorticosterone, progesterone, testosterone, and pregnanediol, which possessed the ability to induce general depression or anesthesia when injected into the peritoneal cavity or when administered through the intravenous or intramuscular route. These results remained largely unrecognized and for a number of years were considered as experiments of purely theoretic and academic interest.

More recently, Laubach, P'an, and Rudel[5] studied a large number of synthetic and natural steroids, seeking one which would not only have an anesthetic action with a good margin of safety and a toxicity no greater than that of anesthetics currently in use; but which would above all be devoid of hormonal activity. In 1955, these investigators discovered 21-hydroxypregnanedione sodium succinate (hydroxydione), initially called P-55 and later Viadril*, a steroid which appears to have the requisites just enumerated.

Tried in various experimental animals by its discoverers. Viadril was found to possess a remarkably high anesthetic potency combined with low toxicity, providing a high therapeutic index. Other advantages of Viadril anesthesia are the low degree of respiratory depression; and the smooth awakening with rapid orientation and minimum postanesthetic depression. It was also demonstrated that Viadril is devoid of estrogenic, androgenic, progestational, corticoid, or gonadotrophic activity.[7]

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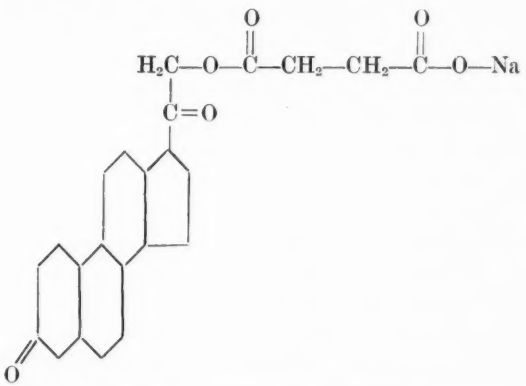
*Viadril brand of hydroxydione is the trade mark applied to 21-hydroxypregnanedione sodium succinate by Chas. Pfizer & Co., Inc.

Viadril is a white crystalline powder, soluble in water, with a molecular weight of 432 and with a pH in solution between 8.5 and 9.8. The chemical structure is as follows:



After the experimental laboratory phase, trials in human subjects began. Viadril was first used in human beings by Gordon and co-workers,[2] who administered Viadril by the intravenous route to 5 schizophrenics and to 3 normal individuals, without any barbiturate or opiate premedication. In the first 2 patients, a small dose of Viadril was used, producing only hypnosis. In the other 6, one of whom received 2 Gm., complete and profound anesthesia was achieved, for a period of time ranging between 20 and 75 minutes. Patients complained of pain along the injected vein when the solution was too concentrated or injection too rapid. No respiratory depression was observed and the awakening was smooth. One unusual effect with Viadril was the fact that, in all 8 patients, the action of the anesthetic was not immediate, but a period of 4 minutes elapsed before anesthesia began to be manifest. Elliott and Sutherland,[1] of the University of California, found an explanation for this interval or period of latency. Studying the consumption of oxygen and of glucose, as well as the cerebral blood flow in the rat's brain in vitro, they observed an interval of 2 hours before the utilization of oxygen was inhibited. It appears that Viadril once injected must be converted to an active metabolite, and that the latency period corresponds to the time necessary for this transformation to occur.

Later, Laborit, Coirault, and associates[3] administered Viadril to 3 patients with delirium tremens. Despite the very high dose required (average of 8 Gm.), administered partly through the intravenous route, partly through the subcutaneous route in conjunction with hyaluronidase, there was no respiratory depression. Signs of delirium tremens disappeared after a few minutes.

A more extensive experience in the human field was that of Murphy, Guadagni, and DeBon,[6] who anesthetized 125 patients for surgical interventions of various types and varying duration. Following premedication, the

patients received, in the operating room, a dose of between 1 and 1.5 Gm. of Viadril by slow injection (5 minutes), and when sleep set in, nitrous oxide-oxygen was administered. Ninety-one patients were intubated, and this maneuver in the majority of cases was performed without curare because of the good relaxation induced by Viadril alone. Most of the patients required no further amounts of steroid, even for operations which lasted for more than 8 hours. Respiratory depression was not encountered, but there was slight diminution in respiratory amplitude. Some patients manifested hypotension, which responded to the usual vasopressor drugs. Three patients experienced pain and venous irritation. The awakening was rapid and uneventful, except for vomiting in a few patients.

Another very complete experimental study is that of Laborit, Huguenard, and co-workers,[4] who concluded that, in animals and in man, Viadril possesses only slight toxicity; induces slow, gradual, and gentle narcosis with rapid awakening; produces no respiratory depression; has no effect upon the sympathetic and parasympathetic systems; affects neither electrolytic equilibrium, nor the cardiovascular system, with the patients maintaining a stable pulse and blood pressure.

I. VIADRIL IN THORACIC SURGERY

Anesthetic techniques for thoracic surgery are many and varied. The one which we generally use at Hospital Sanatorio Nacional Santa Clara consists in the rapid administration of 250 to 300 mg. of thiopental sodium, 6 to 9 mg. of tubocurarine, and oxygen for a few minutes. When the patient is completely relaxed, intubation is performed and the administration of ether and oxygen is immediately started, i.e., anesthesia is maintained with ether, and in contrast to European techniques, thiopental sodium is used only for induction.

In 21 cases of thoracic surgery, we substituted Viadril for thiopental sodium. The types of intervention are listed in Table I.

TABLE I. TYPES OF THORACIC PROCEDURE, 21 PATIENTS

TYPE	NUMBER OF CASES
Lobectomy	13
Pneumonectomy	3
Resection	2
Decortication	1
Pericardectomy	1
Plastic repair	1
Total procedures	21

From reading the experimental and clinical reports of anesthesiologists who were the first to use this steroid, we were aware that the anesthetic action of Viadril is approximately one-half (in the same dose) that of thiopental sodium. Because of this, we decided to use in all patients a single dose of 500 mg., that is, twice the usual dose of thiopental sodium. The age and weight distributions of the 21 patients are summarized in Tables II and III.

TABLE II. AGE DISTRIBUTION

AGE IN YEARS	NUMBER OF CASES
1-10	0
10-20	2
20-30	10
30-40	9

TABLE III. WEIGHT DISTRIBUTION

WEIGHT IN KG.	NUMBER OF CASES
40-50	8
50-60	10
60-70	3

Administration.—Viadril is available in vials of 500 mg., as a white crystalline substance. By injecting into the vial, 20 c.c. of distilled water or isotonic solution, one obtains a 2.5 per cent solution with a very alkaline pH, between 8.5 and 9.8. In order to eliminate irritation of the injected vein sometimes produced by this solution, we added 2 c.c. of 1 per cent procaine to the previously prepared solution. With this precaution, none of the patients operated upon complained of pain, during or after the infusion, even when the steroid was administered rapidly.

Viadril was injected with the same technique and apparatus used for thiopental sodium, and with the patients in the supine position.

Premedication.—Patients received the usual premedication: an oral barbiturate one and one-half hours before intervention and 10 mg. of morphine with 0.25 mg. of scopolamine forty-five minutes before induction. In one patient with bronchial asthma, the barbiturate was replaced by a ganglioplegic and 0.25 mg. of scopolamine.

Induction and Course of Anesthesia.—Induction was extremely gentle, smooth, and gradual; in no case was there any excitation. Intravenous injection of 20 c.c. of Viadril was performed slowly in the first cases, over a period of 5 minutes; in later cases as rapidly as with thiopental sodium. As previously observed by others, Viadril does not have an immediate narcotic action, 4 to 5 minutes elapsing between the moment at which administration ends and that at which the patient no longer responds to questions put to him and at which palpebral and corneal reflexes disappear.

Laryngospasm was not observed nor was there any increase in lacrimal or salivary secretion. Respiratory movements became more superficial, but we never observed apnea. Muscular relaxation is very good, and the patient can be subjected to passive movements without any contractures being observed.

In some patients, once the action of Viadril had become manifest, we tried intubation without curare. This, however, was impossible, and it was always necessary to administer curare in order to obtain sufficient muscular relaxation to intubate the patient without struggling.

No difficulty was encountered in introducing the endobronchial sound of Carlens in 3 patients on whom pneumonectomy was performed.

After intubation a mixture of oxygen and ether was administered. Patients received this without any difficulty and without bronchospasm. Respiratory movements were easily controlled by means of a spiropulsator.

Slight tachycardia without arrhythmia was almost always present at the beginning of Viadril anesthesia. There was also a slight drop in blood pressure. Only 2 patients, who probably had labile cardiocirculatory systems, manifested hypotensive crisis, and then it was necessary to administer a vasopressor, to which the patients responded immediately. Probably hypotension is the result of peripheral vasodilatation, and tachycardia is a compensatory phenomenon. These findings seem to have no relation to the concentration of oxygen in the blood since they were observed also in patients breathing oxygen before and during the administration of Viadril.

Once the patient was in the lateral position, blood transfusion and, in another vein, a dextrose solution containing 3 Gm. of procaine were administered.

Three patients presented urticarial manifestations, at the site of Viadril injection and elsewhere in the body, which disappeared spontaneously in fifteen minutes. The duration of anesthesia in the 21 patients is shown in Table IV.

TABLE IV. DURATION OF ANESTHESIA

HOURS	NUMBER OF CASES
1-2	4
2-3	6
3-4	7
4-5	1
5-6	3

Postoperative Course.—In general, all patients returned to their beds awake, or at least with the laryngeal reflex present. Of the 21 cases, only 2 manifested nausea and vomiting. In the others, the postoperative course was normal. The one observation worthy of mention is that only very small amounts of analgesics were needed by these patients in the first 2 postoperative days, that is, in the most painful period. Of the 21 patients, 10 received, during these 2 days, only minimal doses of sedatives and, in the remainder to whom it was necessary to give morphine, the dose of 10 mg. daily was never exceeded.

Another unusual observation was the fact that 2 patients remained in a state of somnolence and psychomotor depression for 2 days and required no analgesics. They were patients who were well oriented as to time and place, who did what they were requested to do as far as turning over, coughing, and breathing deeply were concerned, and who immediately afterward continued to sleep without complaining of any pain. This postoperative state is very like that seen when tribromoethanol is administered by rectal route.

II. EXPERIMENTAL STUDIES IN HUMAN VOLUNTEERS

Technique.—In 6 patients who volunteered to receive Viadril for purely experimental purposes, we obtained some data which are in accord with observations made by early investigators of Viadril.

In a fasting state and without premedication, these patients received a dose of 500 mg. of Viadril in 2.5 per cent aqueous solution containing 2 c.c. of 1 per cent procaine, administered over a period of one minute. Before the trial, we performed the following tests: partial urinalysis, complete hemogram, analysis of blood glucose, sodium, and potassium, and an electrocardiogram. The latter was performed again when the patients began to sleep, at which time observation of pulse, blood pressure, and respiratory rate at five-minute intervals was also begun.

Three hours after Viadril administration all the tests enumerated above were repeated.

Results.—Following are the electrocardiographic conclusions kindly submitted to us by the cardiologist, Dr. Aníbal Ríos:

"1. Regular sinus tachycardia, explained as sympathetic predominance by virtue of cardiovegetative correlation. The diminution in QT is explained as an increase in frequency.

"2. The second case manifested disappearance of sinus arrhythmia, which can be explained as anesthetic block of environmental stimuli on an organism with neurovegetative disequilibrium.

"3. The lengthening in PR and QRS in the fourth case can be explained as a depression of conduction in the specialized myocardium. The decrease in amplitude of ischemic T waves may be related to the improvement in coronary flow, conforming to the sympathetic hypertonia observed in all cases and corresponding to a pre-established physiopathologic law."

All cases manifested initial tachycardia and slight arterial hypotension.

These phenomena disappeared at the end of 10 to 15 minutes, during which time pulse and pressure returned spontaneously to normal values. Respiration became more superficial, as during physiologic sleep. The urine, hemogram, and blood sugar showed no important changes. The only variation which we found after the experiment in all cases was a slight increase in blood potassium and decrease in blood sodium. These variations did not exceed normal limits.

Patients began to sleep 4 or 5 minutes after the Viadril injection was terminated. The duration of sleep ranged from 15 to 45 minutes. In only one patient was there bradypsychia without loss of consciousness.

We tried Viadril as a "truth serum" but with negative results, since this steroid seems to have an exclusively anesthetic action.

In no case was there any evidence of excitation or vomiting and at the end of the experiment all patients awoke as from spontaneous sleep. They had only one complaint, namely, great hunger.

SUMMARY

Viadril, a synthetic steroid with potent anesthetic properties and a wide margin of safety and without hormonal activity, was administered as a basal anesthetic to 21 patients undergoing thoracic surgery. The compound was

given by intravenous injection following the usual premedication and in conjunction with ether-oxygen. Intubation was performed with tubocurarine. Satisfactory results, with good muscular relaxation and without respiratory depression or laryngospasm, were obtained in a variety of thoracic interventions lasting from 1 to 7 hours. The postoperative course was uneventful, and the small amount of postoperative sedation required was deemed noteworthy.

The compound was also administered experimentally to 6 human volunteers, with careful observations of blood cytology, chemistry, urine, and cardiovascular system. Patients slept from 15 to 45 minutes, awoke naturally, and showed no significant metabolic alterations other than slight tachycardia and hypotension at the beginning of anesthesia.

The author offers grateful acknowledgment to Drs. Celso Jiménez Cadena, Hernán Barona, Aníbal Ríos, Víctor Ortíz, and Hernán Mendoza, for their helpful and distinterested cooperation in this work, and to Charles Pfizer & Co., Inc., for kind cooperation and assistance.

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AIR CONTRAST BRONCHOGRAPHY

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DURING the past five years, this group has utilized the generally accepted method of bronchography by direct injection of radiopaque material into the tracheobronchial tree via an endobronchial catheter. A review of all the bronchograms taken during this period left us with the impression that a modification of technique might result in films that would be of more uniform quality.

Formal discussions with the X-ray Department personnel were instituted with the specific purpose of producing a new modified technique of bronchography that might result in a standardized method of study and eventually produce better bronchograms at a higher ratio than previously afforded.

The following method of study was outlined: (1) review of the literature, (2) choice of radiopaque material, (3) method of instillation of material, and (4) x-ray techniques.

REVIEW OF THE LITERATURE

Schmidt and Donoghue[1] described the various techniques of bronchography dating back to 1905 when Chevalier Jackson injected bismuth oxide powder through a bronchoscope. Diehl[2] presented a review of the various bronchoscopic techniques in use at the present time. He also described a technical modification. Five methods of introducing contrast material were mentioned as being used in bronchography; these are (a) supraglottic or laryngeal, (b) nasal, (c) transglottic, (d) subglottic or intercricoid, and (e) bronchoscopic.

Peele and his associates[3] presented their clinical experience using the water-soluble bronchography compounds with a discussion of contrast materials. Shanks and Kerley[4] in their *Textbook of X-ray Diagnosis* described the techniques of bronchography, divided into transglottic, nasal catheter, and direct injection into the trachea methods.

CHOICE OF BRONCHOGRAPHIC MATERIAL AND METHOD OF INSTILLATION

Until recently, Lipiodol had been utilized exclusively. However, a water-soluble compound (Dionosyl) was obtained and used during the past year. There were fewer problems in its use in that the dye was absorbed quickly and

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did not represent a long duration visible foreign body. We found that when the material was adequately heated (five minutes in a sterilizer) and shaken well until it flowed easily that good quality films were obtained. One of the authors (G. L. H.) suggested that injecting air through the tracheal catheter might produce a contrast type of mucosagram such as is done in gastrointestinal radiography. This was attempted and met with immediate success. The early films using this technique were excellent. Experiments with the amounts of air required to produce good mucosagrams without filling the alveoli showed that between 150 and 200 c.c. of air injected quickly after all of the segments were filled with contrast material sufficed. It seems that the physical properties of Dionosyl in its warm, liquid state are such as to respond to the forcing of air through the catheter by adhering to the bronchial mucosa and producing contrast mucosagrams. This maneuver is repeated when the other side is done. Air is forced into the tracheobronchial tree by rapid injection via the 50 c.c. syringe previously utilized to inject the Dionosyl. There have been no untoward side effects noted.

RADIOGRAPHIC TECHNIQUE

The instillation of the contrast material is done under fluoroscopic observation in the x-ray department. After the tracheobronchial tree has been anesthetized as for bronchoscopy and the catheter (preferably a radiopaque one) has been passed into the trachea, the patient is brought to the fluoroscopic room.

It is customary to do the right side first in any complete bronchogram. The tip of the tube is guided into the right main-stem bronchus and an attempt made to place it at the approximate location of the orifice of the upper lobe bronchus. This can then be checked by the introduction of a small amount of the contrast material which soon enters the proximal portion of the bronchus. If not, the tip can be withdrawn or advanced as desired. The patient is rotated always with the distal portion of the bronchi downward and the table tilted upward and downward as often as necessary to enable the radiopaque material to enter them.

The tube is advanced for the middle and lower lobe bronchi, each in its turn. Rotation and tilting of the table is repeated as often as necessary. At this point, air, for the double contrast mucosagram, is instilled.

Spot films may be taken on 8 × 10 films at any part of the procedure, but usually after the bronchi are well outlined. Our equipment includes an automatic tunnel device with a stationary grid. The factors used for an average chest are 150 milliamperes, 70 kilovolts at .6 sec.

The patient is then sent to the radiographic room and exposures on 14 × 17 films are made in the posteroanterior, right posterior oblique, and right lateral positions. For an average chest of 21 cm. the factors are 300 Ma., 71 kv., 1/30 sec. at 72 inches in the posteroanterior projection. The right posterior oblique projection of such a chest is made using the following factors: 300 Ma.,

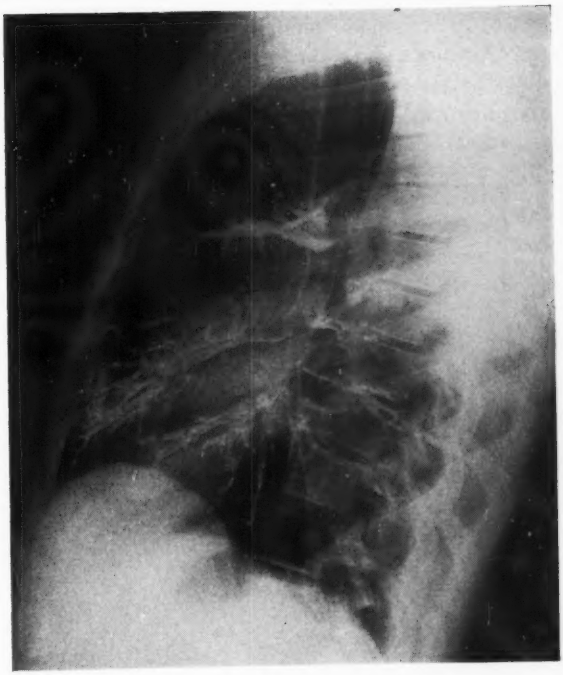

Fig. 1.

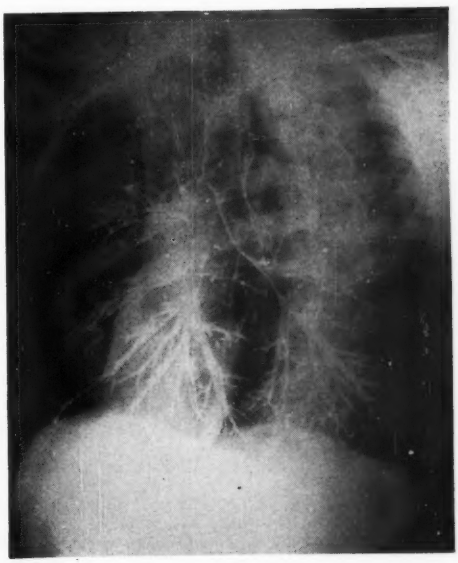

Fig. 2.

81 kv., 1/30 sec., at 72 inches, 45° obliquity. In the lateral exposure, 200 Ma., 71 kv., .35 sec. at 42 inches using a Bucky grid is used. Adjustments of kilovolts are made for differences of chest thickness.

After these three exposures, the patient returns to the fluoroscopic table where the intratracheal catheter is readjusted into the left main-stem bronchus.

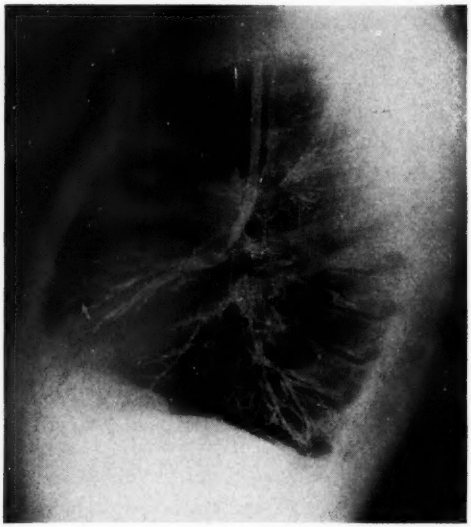

Fig. 3.

Using the same maneuvers, the left bronchi are outlined as on the right. Spot films may be made at this point and then posteroanterior and left posterior oblique views are made, the technical factors being similar to those used for previous comparable views except that in the oblique view a slightly higher kilovolt is used. These are then usually the final roentgenograms.

Figs. 1, 2, and 3 illustrate air contrast bronchography, as just described in the text.

SUMMARY AND CONCLUSIONS

A detailed technique for producing routine high-grade bronchograms has been developed. A method of utilizing air injection into the tracheobronchial tree after filling all segmental bronchi with a water-soluble radiopaque substance (Dionosyl) to produce mucosagrams has been shown to be effective.

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THE EFFECTS OF CORTISONE ON THE HEALING OF THE BRONCHIAL STUMP IN DOGS

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INTRODUCTION

THE observation that there is apparent delay in wound healing in patients who are receiving cortisone or ACTH has led to numerous experiments to determine the effects of these agents on tissue repair.

Goodman and Gilman[9] state that hydrocortisone interferes with formation of granulation tissue, fibroblasts, ground substance, and vascularization, resulting in delay in the healing of wounds. These conclusions are in accord with those of Lukens.[10] Though the inhibitory effect of cortisone varies considerably from species to species, the response of the individual animal is in part determined by its physical condition. Protein-depleted rabbits have been shown to require smaller doses of cortisone to depress fibroplasia than do healthy rabbits. A similarity has been noted in the healing deficiencies of cortisone treatment and those of scurvy. However, in rabbits receiving both cortisone and ascorbic acid, wounds showed neither histologic nor tensile strength differences when compared with those in rabbits receiving only cortisone.[8] The degree of impaired wound healing appeared directly related to the dosage of cortisone employed, according to Meadows and Prudden,[11] in their studies on rats. On the basis of experiments with uretero-intestinal anastomoses in dogs, Baker and his co-workers advanced the opinion that cortisone causes a reduction in the increased fibroplasia seen in infected wounds.

That wounds in rats show delayed healing under the influence of cortisone is further attested by the work of Taubenhaus' group,[15] which injected turpentine subcutaneously in its animals. Alrich and others obtained evidence of delayed healing of abdominal wounds in cortisone-treated rats.[1] Using rabbits, Ragan and associates observed retardation in the development of all elements of connective tissue in skin wounds.[13] They noted that experimental fracture of the femur in cortisone-treated rabbits was followed by a great delay in the healing and absorption of the hematoma.[4] They demonstrated further that the degree of inhibition of healing in cutaneous wounds of rabbits was dependent on the dose of cortisone employed.[14] Though heavy doses of cortisone were

From the Research Laboratory, the Surgical and Laboratory Services of the Thayer Veterans Administration Hospital and the Departments of Surgery and Pathology of the Vanderbilt University School of Medicine.

The cortisone used in this experiment was supplied by Dr. Elmer Alpert, Merck and Company, Rahway, N. J.

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used, Clayton was unable to show reduced healing of wounds in guinea pigs.[5] Wrenn and his co-workers, working with dogs, found that the disruptive force required for sutured tendons was less in cortisone-treated dogs than with control animals.[16] Excessive formation of peritendinous fibrous tissue and regeneration of tendon sheaths were also seen to be inhibited in the cortisone-treated dogs. Muller and his associates found no significant difference in the disruptive strength of aortic anastomoses (without graft) in cortisone-treated dogs as compared with control dogs,[12] but Barberio and co-workers reported evidence of impaired healing of aortic grafts in dogs receiving cortisone.[3] Cole's group discovered no gross or microscopic differences in skin healing of dogs receiving relatively small doses of cortisone as compared to control animals.[6] A mild and variable inhibitory effect on fibrous tissue regeneration in wounds of human beings receiving cortisone was reported by DeKleine, though this effect was not sufficient of itself to control keloid recurrences.[7]

Recently we had to resort to the use of cortisone in the immediate postoperative period following a pulmonary resection. Although the patient's convalescence was uneventful, the case served to stimulate our interest in the effect of cortisone on the healing of the bronchial stump.

METHOD

Pneumonectomy was done on 41 dogs of both sexes, which were grouped as follows:

Group I—sacrificed at 14 days after pneumonectomy.
- 9 control dogs.
- 8 test dogs, receiving 4 mg. of cortisone per kilogram of body weight per day intramuscularly from the operative day until sacrifice.

Group II—sacrificed at 21 days after pneumonectomy.
- 9 control dogs.
- 5 test dogs, receiving 8 mg. of cortisone per kilogram of body weight per day intramuscularly from the operative day until sacrifice.

Group III—sacrificed at 21 days after pneumonectomy.
- 9 control dogs (same animals as for Group I).
- 10 test dogs, receiving 4 mg. of cortisone per kilogram of body weight per day intramuscularly from three days prior to operation until sacrifice.

Using intravenous Nembutal, and endotracheal airway, and a mechanical respirator, a left pneumonectomy was performed on each animal. The cut end of the left main-stem bronchus was closed by interrupted 4-0 cotton sutures and the bronchial stump was covered by closure of the mediastinal pleura. Penicillin and streptomycin were administered for five days following surgery. The dogs were kept in laboratory cages and were fed meat scraps and canned dog food. Sacrifice was effected by Nembutal anesthesia and electrocution. The

specimens were examined grossly for evidence of bronchopleural fistula, and were then removed for pressure tests. A catheter with an inflatable cuff was placed in the trachea, connected to a pressure bulb and mercury manometer, and the right main-stem bronchus was clamped. Immersing the specimens in water, the pressure at the bronchial suture line was raised to a minimum of 12 cm. of mercury, or until a leak was produced. After pressure tests, tissue was excised from each bronchial stump for microscopic study.

Failure of bronchial stump healing was established by the finding of an obvious bronchopleural fistula or air leakage at the bronchial stump at pressures below 12 cm. of mercury during pressure tests. Empyema with an intact bronchial stump, or stump leakage at pressures greater than 12 cm. of mercury were not considered due to defective bronchial stump healing.

RESULTS

Of 18 control animals, one died on the eighth postoperative day with a bronchopleural fistula and the other 17 survived to sacrifice. With pressure tests, the bronchial stump of one of the control dogs of Group I leaked at 12 cm. of mercury pressure. The bronchial stumps of the remaining control dogs of all groups withstood pressures in excess of 12 cm. of mercury without leakage.

All the cortisone-treated dogs of Group I survived until sacrificed, and the bronchial stumps of all 8 withstood pressures of 12 cm. of mercury or more. Thus, there was one stump failure among all the control dogs and no stump failure among the test dogs of Group I.

The 5 cortisone-treated dogs of Group II survived until sacrificed. During electrocution, 2 of these animals developed subcutaneous and mediastinal emphysema; and on pressure testing, the bronchial stump of one of the dogs so afflicted showed a leak at the suture line at zero pressure. No leaks occurred in the bronchial stumps of the remaining 4 dogs when subjected to pressures of 12 cm. of mercury or greater. Result: one stump failure in the cortisone-treated dogs of Group II.

Of the 10 cortisone-treated dogs in Group III, there were two failures of stump healing. One dog died on the tenth postoperative day with a bronchopleural fistula. A second animal survived to sacrifice, but the bronchial stump leaked at a pressure of 5 cm. of mercury. Two other dogs showed complications which were not attributable to deficient healing of the bronchial stump. One of these dogs died on the fourteenth postoperative day with empyema and an intact bronchial stump which was tested to a pressure of 26 cm. of mercury before leaking. An additional dog was suspected clinically of having a bronchopleural fistula, but survived to sacrifice, and the bronchial stump in this instance withstood a pressure of 48 cm. of mercury before leakage occurred. The bronchial stumps of the remaining animals in Group III withstood pressures in excess of 12 cm. of mercury without leakage. These results are summarized in Table I.

TABLE I. FAILURE OF HEALING OF BRONCHIAL STUMPS WITH VARYING CORTISONE DOSAGES

	NUMBER OF DOGS	CORTISONE DOSAGE	NUMBER OF DOGS WITH BRONCHOPLEURAL FISTULA
GROUP I	8	4 mg. per kilogram of body weight per day for 14 days postoperatively	0
GROUP II	5	8 mg. per kilogram of body weight per day for 21 days postoperatively	1
GROUP III	10	4 mg. per kilogram of body weight per day for 3 days before and 21 days after operation	2
CONTROLS	18	None	1

Comment.—From a statistical analysis of results derived from the binomial expansion, based on the probability tables of Warwick, it is unlikely that the proportion of stump failures in any of these groups is different from zero.* Of the 41 animals used, only 2 dogs developing bronchopleural fistula showed gross evidence of separation of the sutured edges of the bronchus. A third bronchus had a barely visible fistula between the sutures, and the fourth failure of stump healing was established because of air leakage at 5 cm. mercury pressure during tests.

MICROSCOPIC STUDY

Microscopic sections of all bronchial stumps were stained with hematoxylin and eosin, and with the Mallory connective tissue stain. Since the dogs used in the experiment varied considerably in size, an allowance was made for the size differences in comparing the sections. The number of fibroblasts present, the amount of collagen formation, the degree of foreign-body reaction and the vascularity were the main points of comparison in evaluating the sections.

Group I. Controls.—Healing of the bronchial stumps in these animals was generally well advanced and fibroblasts were present in abundance. In those instances where a pleuritis was evident, there was a considerable degree of pleural organization. Adequate maturity of the fibroblasts was reflected by the liberal amount and compactness of the collagen fibers. Foreign-body giant cells were common in the areas of suture, and in some places their numbers were striking. Vascularity of the bronchial stumps was of the expected magnitude.

Group I. Cortisone-Treated Dogs.—Noticeable fewer fibroblasts were observed in the healing bronchial stumps of these dogs. Pleuritis failed to incite the degree of organization seen in the control animals. Though no significant differences were detected in the characteristics of the individual fibroblasts of the control versus the cortisone-treated animals, collagen deposition was restricted to an even greater extent in the latter than the numbers of fibroblasts would indicate. Foreign-body giant cells were fairly numerous about the suture material, but the reaction was not as prominent as with the controls. Vascular ingrowth was questionably less in the cortisone-treated dogs, but a

*We are indebted to Mrs. Con O. T. Ball of the Radioisotope Service of Thayer V. A. Hospital for the statistical analysis of the data in this study.

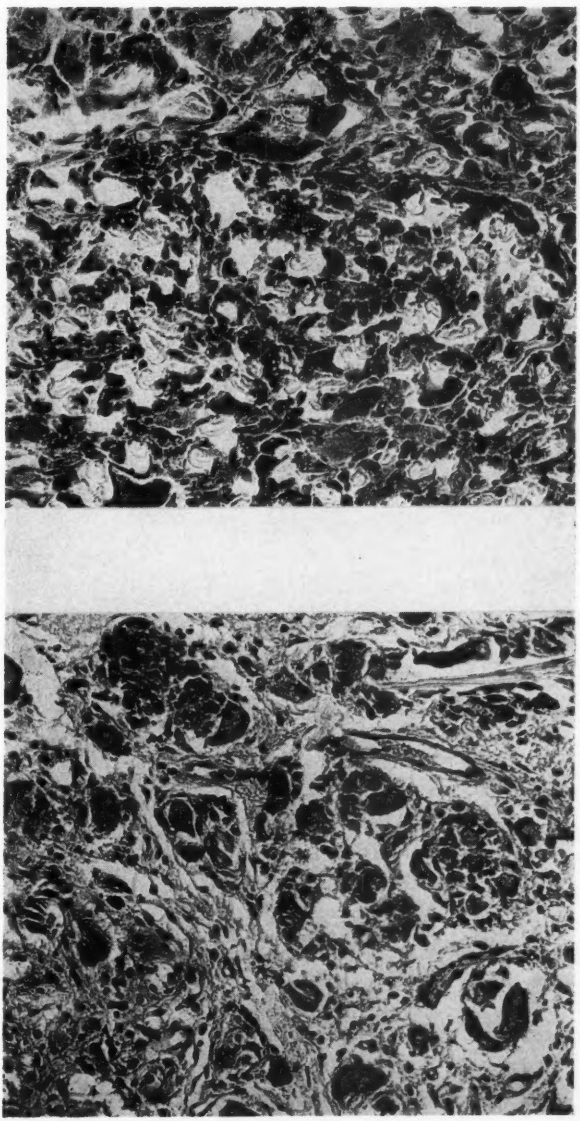

Fig. 1.—Comparable photomicrograph fields illustrate the intensity of foreign body reaction in the control animal (above) with the diminished response in the cortisone-treated animal (below). (Hematoxylin and eosin; × 200.)

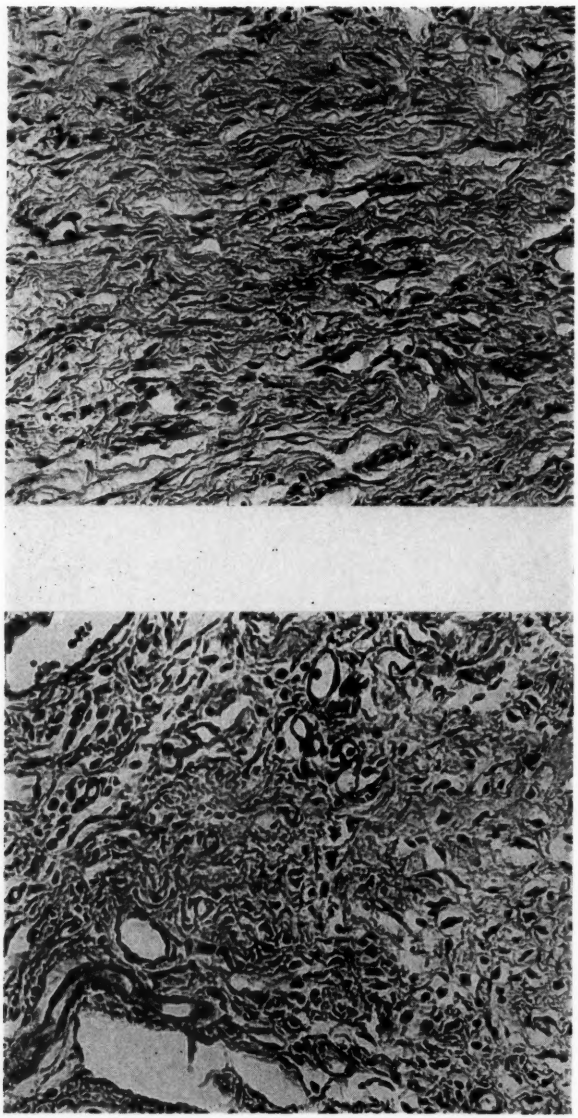

Fig. 2.—A microscopic comparison of fibroplasia in a control dog (above) and a cortisone-treated dog (below) shows the healing tissue of the latter to be distinctly more vascular, but less cellular, and with less abundant production of fibrils. (Hematoxylin and eosin; × 200.)

lingering hyperemia was apparent that was not seen in the controls. The degree of inflammation, as judged by collections of leukocytes, was approximately the same for both categories.

Groups II and III.—Comparing the controls with the cortisone-treated animals in Groups II and III, the results were essentially the same as for Group I. In each group, comparisons of controls with cortisone-treated animals were confined to the group in question. There was no attempt to quantitate the results shown with different cortisone doses or sacrifice times.

Comment.—A reduction in the number and activity of the fibroblasts was the most striking effect produced with cortisone. There was also a slight decrease in the foreign-body response to the suture material in the cortisone-treated animals. Only a suggestion of decreased vascularity of the stump was noted in the cortisone-treated dogs, and the inflammatory response as determined by collections of leukocytes was similar to that of the controls.

SUMMARY AND CONCLUSIONS

Moderate inhibition of healing of bronchial stumps was observed in a series of dogs treated with cortisone. The evidence to support this conclusion was essentially of a microscopic nature.

On the basis of the clinical course and the results of testing the bronchial stumps at autopsy, it is apparent that there is little danger of deficient bronchial stump healing in dogs treated postoperatively with cortisone in dosages comparable to those used in man. It would seem safe to give full therapeutic doses of cortisone for relatively short periods of time in the postoperative period after pulmonary resection in human beings. However, because of the microscopic changes seen in this series, there is some doubt that it is entirely safe to do an elective pulmonary resection on a patient who has been treated with cortisone for a protracted period prior to operation and who will require prolonged cortisone therapy postoperatively.

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CHORDOMA OF THE THORACIC SPINE APPEARING AS A MEDIASTINAL TUMOR

Report of Two Cases

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EVEN in the most exhaustive reviews of mediastinal tumors, chordoma is not mentioned as a possibility among posterior mediastinal tumors. Two cases reported below show that the possibility of chordoma should be taken into consideration when a posterior mediastinal tumor with vertebral destruction is detected radiologically.

CASE REPORTS

Case 1.—F. P., a 43-year-old man, was seen in the Institute of Oncology in Warsaw in November, 1948. Four years prior to examination he had received x-ray treatment in the Institute because of pains in the chest which had been present, at that time, for about a year. (All records were lost during the war.) Three months after treatment he felt quite well; the pains were absent. Four years later symptoms reappeared and gradually increased.

On admission, physical examination did not reveal any abnormality. Radiologic examination of the chest revealed a tumor of the upper posterior mediastinum, well demarcated and situated more on the left side but visible also to the right of the mediastinal shadow (Figs. 1 and 2). He was directed to the chest surgical unit with the tentative diagnosis of a tumor of neural origin. However, he refused surgical treatment and reappeared at the Institute. X-ray examination of the spine showed destruction of the anterior parts of the thoracic vertebra, T_3 and T_4 (Fig. 3). The neural arches of these vertebrae did not show destructive changes at that time. Since previous x-ray treatment had resulted in alleviation of symptoms, he again received a course of x-ray treatment. Four fields (two lateral, anterior, and posterior) were irradiated, 1,000 to 1,400 r being given to each. Pain in the chest regressed again but the radiologic appearance did not change. Six months later he was brought to the Institute as an emergency case with paralysis of the lower limbs. Neurological examination revealed flaccid paralysis of both lower extremities and sensory disturbances to the level of the fourth thoracic vertebra. The radiologic examination of the chest showed marked increase of the tumor size (Fig. 4). Examination of the spine showed slight right-sided scoliosis, more extensive destruction of anterior parts of the bodies of T_4, T_3, and T_5, with partial collapse of T_4 (Fig. 5), and somewhat irregular outlines of the intervertebral foramina between T_4 and T_3 and T_4 and T_5.

On laminectomy (W. Rudowski), compression of the spinal cord with its displacement to the right side was seen. A pinkish-gray tumor was growing into the spinal canal, infiltrating the spine and long muscles of the back. The intraspinal part of the tumor was removed; the spinal cord was returned to its normal position and began to pulsate. Two days after the operation, the patient died, most probably from acute circulatory failure. No post-mortem examination was performed. Histologic examination of the part of the tumor removed during operation showed structure typical of a chordoma.

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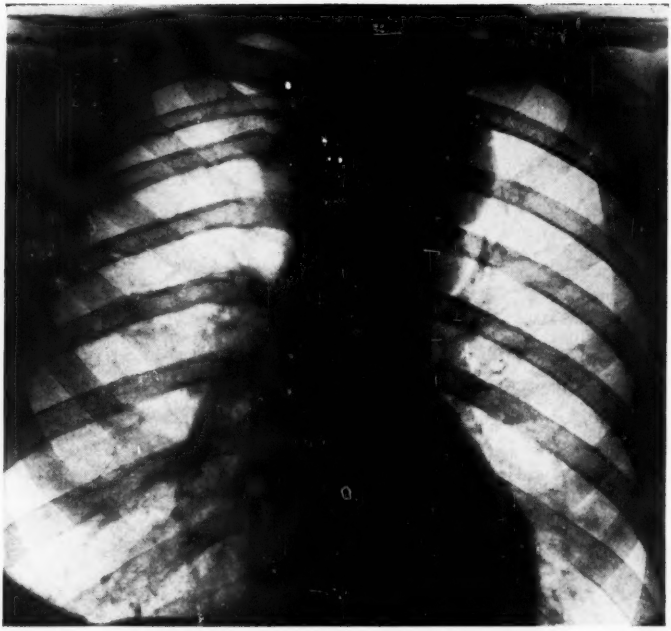

Fig. 1.—Case 1. Mediastinal tumor appearing on both sides of the median shadow.

Fig. 2.—Case 1. Lateral radiograph showing the posterior position of the tumor.

Fig. 3.—Case 1. Oblique radiograph of the thoracic spine showing destruction of the frontal part of T_3 and T_4.

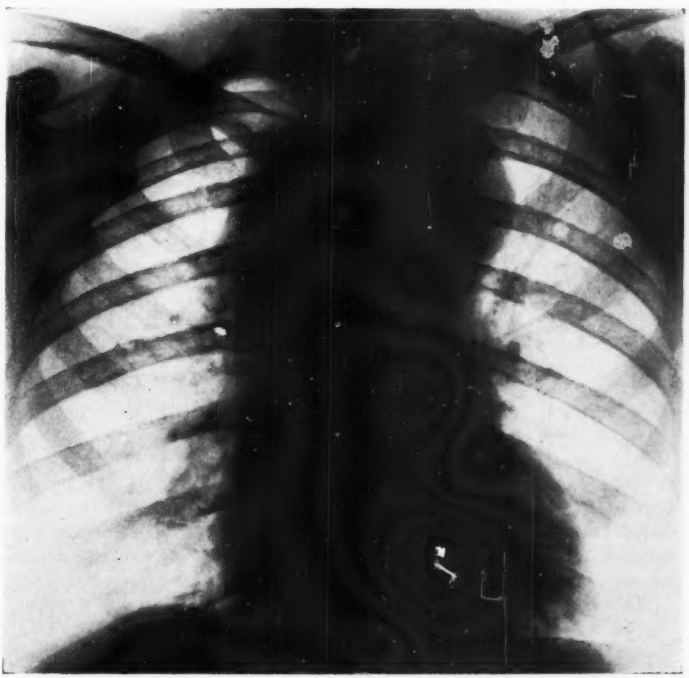

Fig. 4.—Case 1. Mediastinal tumor after six months' time.

Fig. 5.—Case 1. Oblique radiograph of the thoracic spine showing further destruction of vertebral bodies (T_3, T_4, and T_5).

Case 2.—W. S., a 37-year-old man, was seen in the Institute in October, 1953, complaining of pains in the chest located in the lower left scapular area. Clinically, no abnormalities were detected. Radiologic examination revealed a sharply outlined upper mediastinal tumor visible on both sides of the median shadow, but more prominent to the left (Fig. 6), and situated quite posteriorly. The tentative diagnosis of posterior mediastinal tumor, probably of neural origin, was made, but the patient was not admitted to the hospital since he decidedly refused operative treatment. He reappeared a month later complaining of pulmonary hemorrhages, disturbances of gait, and sensory changes in the lower limbs. Radiologically, a slight increase of tumor size was noted. Radiologic examination of the spine showed pathologic fracture and flattening of the body of the T_4 and the destruction of the left parts of the neural arch of this vertebra (Figs. 7 and 8). Thus, the mediastinal tumor was described as in some way enveloping the vertebra from at least three sides as it entered the spinal canal.

Neurological examination showed spastic paresis of both lower limbs and sensory disturbances of all kinds accentuated on the left side to the level of the second rib; disturbances of tactile and temperature sense was demonstrated on the right side.

Abdominal reflexes were abolished, tendon and periosteal reflexes increased in both limbs and were clonic in character.

On laminectomy (W. Rudowski) of the third through the sixth vertebrae, an intraspinal tumor was present at the level of T_4, with a diameter of 2.5 by 1.5 cm., growing into the canal from the intervertebral space. The spinal cord was displaced to the right and compressed. After removal of the intraspinal part of the tumor, bone destruction of the fourth thoracic vertebra was macroscopically noted. After the operation, the patient's neurological condition gradually improved. Sensory disturbances disappeared but paresis of the limbs persisted. He was discharged at the wish of the family. At home, his condition improved rapidly. He began to walk and in two months all discomfort disappeared.

Pathologic examination of the part of the tumor removed showed chondroma with areas resembling chordal tissue present.

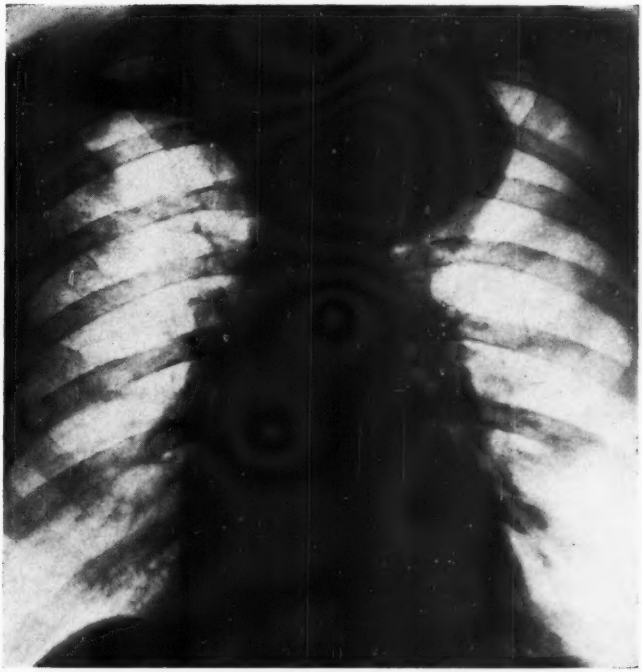

Fig. 6.—Case 2. Mediastinal tumor appearing on both sides of the median shadow, more prominent to the left.

DISCUSSION

1. It is generally accepted that chordomata originate in the remnants of the embryonal notochordium. Horwitz,[10] examining a large number of human embryos, found that heterotopic remnants of notochordium are most often present in those sites favored by chordomata. It is supposed that heterotopic remnants are more liable to undergo neoplastic metaplasia; that would explain the predominance of some situations.

2. Chordomata of the spine are most often situated in the sacrococcygeal region. Other spinal localizations are very rare and the thoracic region is quite exceptional. Faust, Gilmore, and Mudgett[6] collected all previous reports of chordomata, totaling 352 cases, and only 34 were in sectors of the spine other than sacral and coccygeal. Baker and Coley[1] collected reports of 13 more cases and described one case situated in the lumbar region. Among Dahlin and Mac Carty's 59 cases, 11 were spinal (none including sacrococcygeal).[5] Sennet[15] described 11 cases, among them 3 were cervical and 3 were lumbar. Thus, the total number of vertebral chordomata amounts to 64 cases, perhaps a few more.

Location in the thoracic region is most unusual. Mabrey[11] and Hanson[8] described a tumor located in the area of T_7, with destruction of the

Fig. 7.—Case 2. Oblique radiograph of the thoracic spine showing destruction and pathologic fracture of T_3.

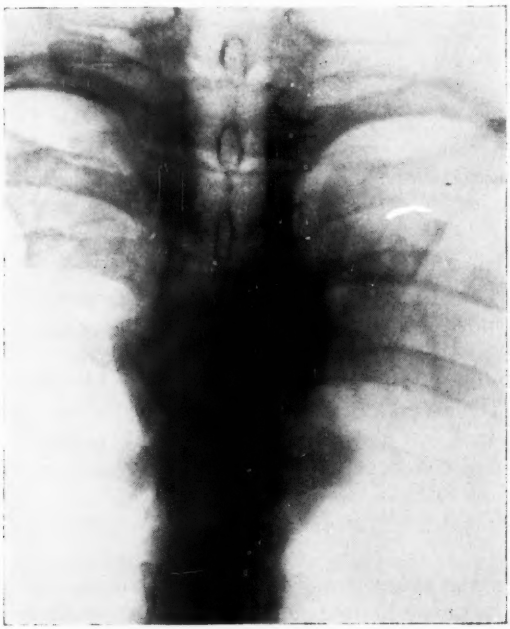

Fig. 8.—Case 2. Anteroposterior radiograph of the thoracic spine showing effacement of the left part of the neural arches of T_3.

neural arches. Willis had seen one localized in T_1 and T_2, posteriorly situated also. In Wood and Himadi's[17] case, T_{11} and T_{12} were affected. One of the tumors reported by Dahlin and Mac Carty was situated in the eleventh thoracic vertebra causing compression of the spinal cord.

This short list, even if incomplete, shows how unusual the thoracic location of chordomata is and explains to some extent why the chordoma has so far not been described as a mediastinal tumor.

3. The radiologic appearance of both of our tumors are characteristic enough to allow some conclusions concerning the radiologic differential diagnosis (J. Buraczewski, and W. Rudowski). Chordoma of the vertebrae, in the more advanced stages, appears as a tumor mass enveloping, so to say, the body of the vertebra from at least three sides and may be visible on both sides of the spinal column and posteriorly when it causes destruction of the neural arches or enters the spinal canal. It causes the destruction of vertebral bodies with their eventual collapse. The drawing (Fig. 9) represents schematically how it differs from the usual hourglass tumors of neural origin (Fig. 10).

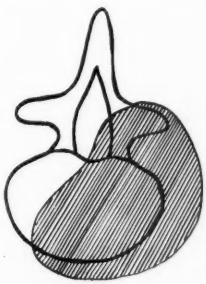

Fig. 9.

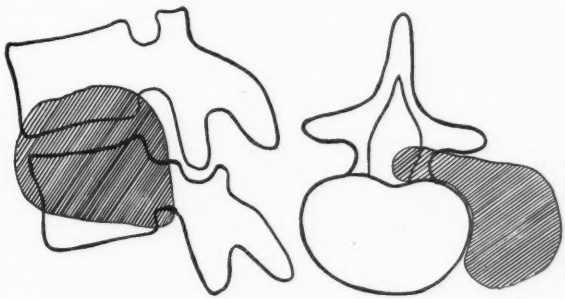

Fig. 10.

Fig. 9.—Chordoma of the vertebrae schematically represented.
Fig. 10.—Drawing presenting schematically common appearance of the hourglass tumor of neural origin.

Such radiologic appearances could perhaps be attributed to other malignant tumors of the spine, particularly a primary tumor of the bone, but these are probably as unusual as chordomata with the exception of multiple myeloma involving the spine. Such radiologic appearances as just described, when supported by clinical evidence of long-lasting symptoms with small static disturbances, especially when more than one vertebra is involved, should allow the tentative diagnosis of chordoma. The thoracic spine perhaps presents better conditions for viewing all these radiologic changes than the lumbar sector does.

It should be mentioned that the same description of a tumor enveloping the vertebral body from at least three sides, including the posterior wall, may be applied to the sacrococcygeal chordomata, in which case additional information can be gained by vaginal or rectal examination.

Fig. 11.

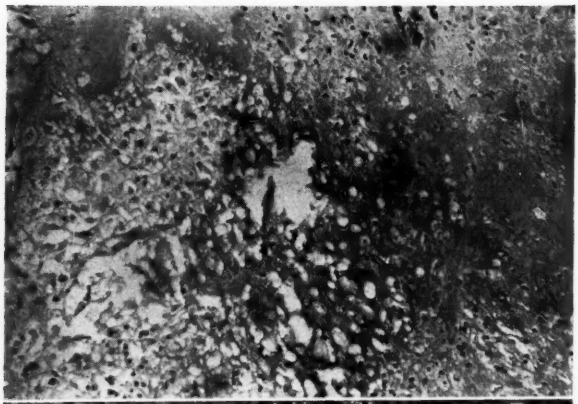

Fig. 12.

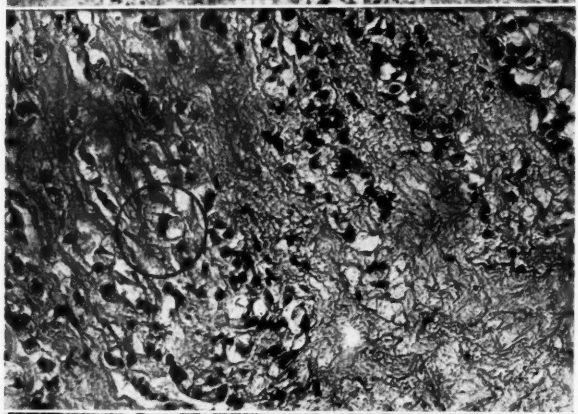

Fig. 13.

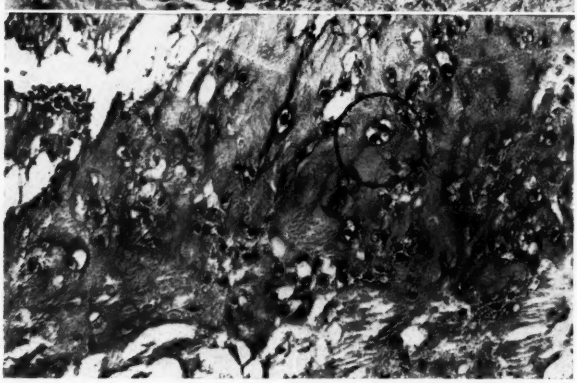

Fig. 11.—Case 1. Photomicrograph of the more differentiated area of the tumor composed of the intercellular substance with the clear, polyhedral chordal-like cells loosely applied one to another. (Magnification ×120; reduced ⅓.)

Fig. 12.—Case 1. The neoplastic cells predominantly in cords separated by the mucoid matrix of the tumor itself. The typical "physaliphorous" cell is marked with circle. (Magnification ×240, reduced ⅓.)

Fig. 13.—Case 2. In areas of the mucus formation the solitary "physaliphorous" cells are visible. The typical cell is marked with circle. (Magnification ×240; reduced ⅓.)

On the strength of such clinical and radiologic evidence, we diagnosed the second of our cases as chordoma and two other tumors, one thoracic and one lumbar, in which, however, it was not possible to confirm the diagnosis by operation and subsequent pathologic examination.

4. Some doubt may be expressed as to the nature of the tumor in our second case in which the histologic examination revealed chondromatous tissue with some inclusions resembling chordoma. But the case, in all its aspects,

Fig. 14.

Fig. 15.

Fig. 14.—Case 2. In the lower right corner the chordal cells are present in the borderline between the connective tissue and the mucoid masses. (Magnification ×240; reduced ⅓.)

Fig. 15.—Case 2. Great "physaliphorous" cells with characteristic vacuolation situated near the basal line in the periphery of the tumor and floated in the mucoid matrix. (Magnification ×240; reduced ⅓.)

seems to give sufficient evidence for the diagnosis of chordoma. Chondroma with such feature sas this case presents is difficult to imagine. There is also a striking similarity to our first case which presented the typical structure

of chordoma. It is well known that chondromatous tissue, or tissue histologically strongly resembling cartilage, is often present in chordomata but its significance is not yet quite clear.

5. It is necessary to add chordoma as a possibility in the list of posterior mediastinal tumors.

SUMMARY

Two posterior mediastinal tumors presenting clinically the syndrome of spinal cord compression are described. Radiologically, both these cases presented appearances of upper posterior mediastinal tumors visible on both sides of the median shadow with destruction of the bodies of the thoracic vertebrae as well as of the neural arches.

In both cases laminectomy was performed and the intraspinal part of the tumor removed. Histologically, Case 1 presented the typical appearances of chordoma. The tumor of Case 2 was composed of chondromatous tissue with inclusions of tissue resembling chordoma.

Radiologic and clinical evidence allowing the tentative diagnosis of spinal chordoma is discussed.

Chordoma should be considered as a possibility when the differential diagnosis of posterior mediastinal tumors is considered.

The authors wish to express thanks to Prof. Dr. J. Laskowski from the Oncological Institute in Warsaw for kind permission to publish microphotographs and to Dr. Dabska for preparation of prints.

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PLEUROPULMONARY COMPLICATIONS OF TULAREMIA: TWO REPORTS OF TULAREMIC LUNG ABSCESSES

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EVANSTON, ILL., AND LITTLE ROCK, ARK.

A SEARCH of the literature has revealed that only 4 cases of tularemic lung abscesses have been reported. Three of those were found at autopsy. We present herein 2 additional cases, in which the patients survived as a result of proper management. A residual cavity was treated in one by segmental resection.

CASE REPORTS

CASE 1.—S. H., a 36-year-old Negro man, entered the hospital on Dec. 20, 1949. He had been well until a sudden onset of chills, fever, and malaise five days previously. On Dec. 17, 1949, he had been admitted to another hospital where he had received penicillin every three hours. On December 18, he began to cough and raise scant mucopurulent sputum. The patient stated that one week prior to the onset of the illness he had gone hunting and had shot several rabbits and squirrels. These were dressed but no cutaneous lesions or lymphadenopathy developed.

Past History.—The patient had had a penile ulcer in 1935 treated weekly, for an indefinite period, by intramuscular injections. In 1944, examination of the cerebrospinal fluid allegedly was reported to be negative. He had been a resident of Imperial Valley in California in 1948 without illness.

Physical Examination.—The patient was well developed and well nourished but acutely ill. Loud rhonchi and râles were heard over the anterolateral aspect of the right chest. The spleen was felt only the first two days of hospitalization. Physical examination was otherwise negative.

Laboratory Data.—Hemoglobin, 12.4 Gm.; white blood cell count 10,500, with a normal differential; sedimentation rate, 25 mm. per hour. Urinalysis was normal except for one plus albumin, many granular casts, and 20 to 25 white blood cells per high-power field. A serological test for syphilis was negative. Sputum cultures for acid-fast bacilli and fungi were negative. Agglutination tests, including that for tularemia, were negative on admission. Blood cultures were sterile. However, repeat agglutination test for tularemia on Dec. 24, 1949, was positive (1:320) and increased to 1:1280 on Jan. 6, 1950.

Roentgenologic Data.—Thoracic roentgenogram on Dec. 21, 1949, showed a 3 by 6 cm. area of pneumonia in the anterior portion of the right lower lobe and a 1.5 cm. nodule in the right first interspace. Subsequent serial roentgenograms revealed development of a 2 by 4 cm. cavity in a lesion of the right lower lobe (Fig. 1). Planigrams (Jan. 3, 1950) revealed a definite cavity at the 10 cm. level. By January 17, infiltration of the right lower lobe and size of the cavity were regressing. On February 20, only linear fibrosis remained. The 1.5 cm. nodule in the right first interspace remained unchanged.

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Course in the Hospital.—The patient's admission temperature was 104° F. He was started on Aureomycin, one gram daily, on Dec. 21, 1949. Within a few days, the temperature was normal. Cough and expectoration of sputum rapidly subsided. Tuberculin, coccidioidin, and histoplasmin skin tests were positive. Tularemia was strongly suspected because of the history of handling rabbits. With the development of a high agglutination titer for tularemia, the diagnosis was established. Since the patient had been in an area endemic for coccidioidomycosis, the small lesion in the right upper lung field was suspected of being either coccidioidomycosis or tuberculosis.

At the follow-up examinations on June 15, 1951, and June 11, 1952, the patient was asymptomatic and the thoracic roentgenograms taken demonstrated no change from those of February, 1950.

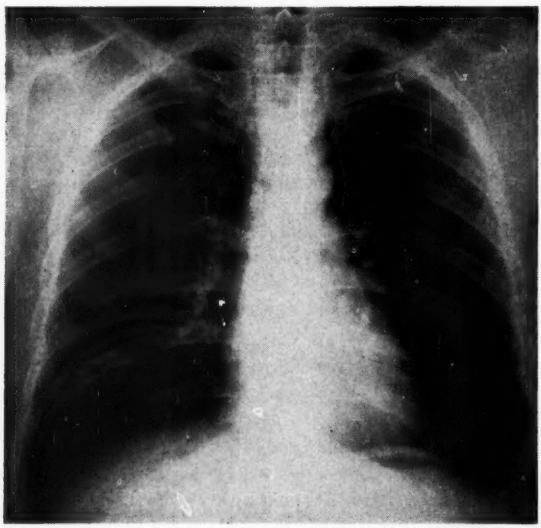
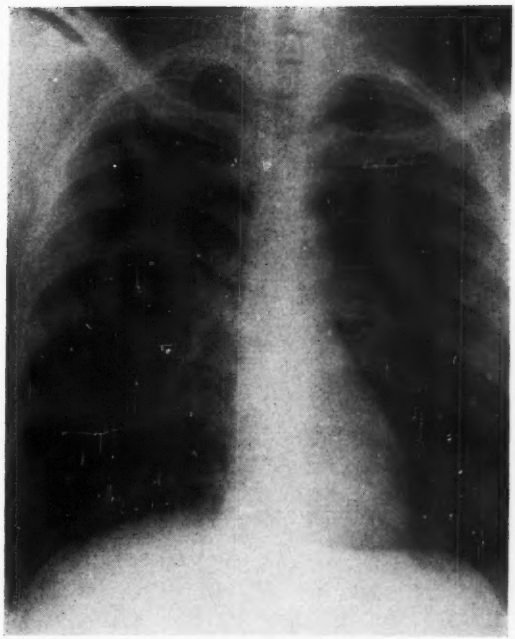


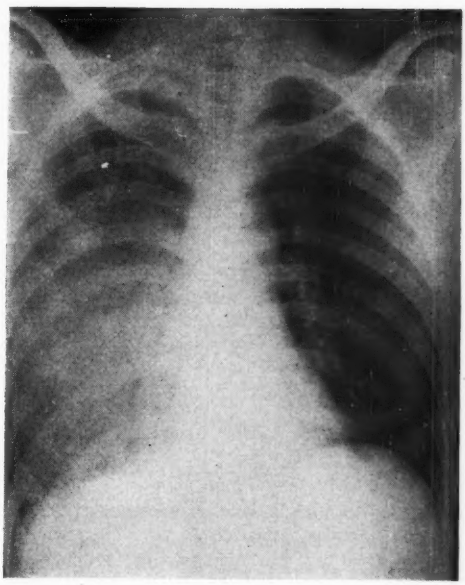
Fig. 1.—Case 1. Roentgenogram of Dec. 30, 1949, showing lung abscess of right lower lobe.

CASE 2.—J. A., a 44-year-old unemployed Negro farmer, was readmitted to the hospital on Aug. 25, 1950, in a delirious state. The history was obtained from relatives. The patient was allegedly well until eleven days previously when he complained of malaise and being chilled. After going to bed he began to have a very severe right-sided headache which persisted for thirty-six hours. An accompanying elevation of temperature to 104° F. continued for a week. Insomnia was also present. Other complaints were right lower chest pain, loss of strength, and anorexia. On August 22, a physician made a diagnosis of pneumonia and prescribed two injections of penicillin and "sulfa" tablets. The patient developed hiccups and subsequently took only small amounts of fluid by mouth. It was also reported that icteric sclera and dark urine were noted on the first day of the illness. Inquiry failed to reveal significant epidemiologic information although a past history of chronic alcoholism and treated syphilis and gonorrhea were established.

Physical Examination.—The admission temperature was 102° F.; pulse rate, 120; respiration, 30. The patient appeared well developed, slender, extremely dehydrated, critically ill with frequent hiccups (fifteen per minute). The skin was hot, dry, and free from rash, ulcer, or petechiae with generalized hyperesthesia. Definite nuchal rigidity. Sclera



A.



B.

Fig. 2.—Case 2. *A*, Normal thoracic roentgenogram of previous admission (June 6, 1949). *B*, Admission roentgenogram (Aug. 24, 1950) demonstrating right pleural effusion which was loculated posteriorly and displaced the lower lobe anteriorly. The upper and middle lobes were clear but a rather extensive consolidation of the apex of the lower lobe was suspected.

icteric and the eyes were sensitive to light. There were dullness to percussion and diminished breath sounds over the right lung below the scapular angle. The abdomen was slightly distended with the liver palpable 6 cm. below the right costal margin. There was no splenomegaly or lymphadenopathy.

Laboratory Data.—Hemoglobin, 15.6 Gm.; erythrocytes, 5.42 million; leukocytes, 10,450 with 85 per cent polymorphonuclear leukocytes and 15 per cent lymphocytes. Urinalysis showed marked albuminuria with numerous granular casts and 12 to 15 erythrocytes per high-power field. Serological tests for syphilis were positive. Serum urea nitrogen, 48 mg. per cent; glucose, 89 mg. per cent; carbon dioxide combining power, 13.1 meq. per liter; chlorides, 107 meq. per liter; and total proteins, 5.4 mg. per cent with albumin 2.8 mg. per cent and globulin 2.6 mg. per cent. Icteric index, 61 units. Van den Bergh's test—direct, 10.2 mg. per cent; indirect, 11.5 mg. per cent. Prothrombin time, 46 per cent of normal. Sedimentation rate, (corrected), 26 mm. Cerebrospinal fluid, clear yellow, 95 erythrocytes, and 450 leukocytes per cubic millimeter; glucose content, 33 mg. per cent; protein, 186 mg. per cent; complement fixation, negative. Sanguineous pleural fluid, 76,300 erythrocytes and 6,900 leukocytes per cubic centimeter with 29 polymorphonuclear leukocytes, 66 lymphocytes and 5 monocytes; chemical analysis, 113 mg. per cent of glucose and 5.3 mg. per cent of protein; acid-fast bacilli, none on smear. Initial serum agglutination titers: paratyphoid A, 1:20; paratyphoid B, 1:80; *Brucella abortus* and Weil-Felix reaction, negative; tularemia, 1:20. Blood culture was sterile. Subsequent antitularense agglutination titers: Aug. 30, 1:40; Sept. 15, 1:160; Sept. 28, 1:320; Jan. 25, 1951, 1:1280. Sputum examinations were negative for acid-fast bacilli.

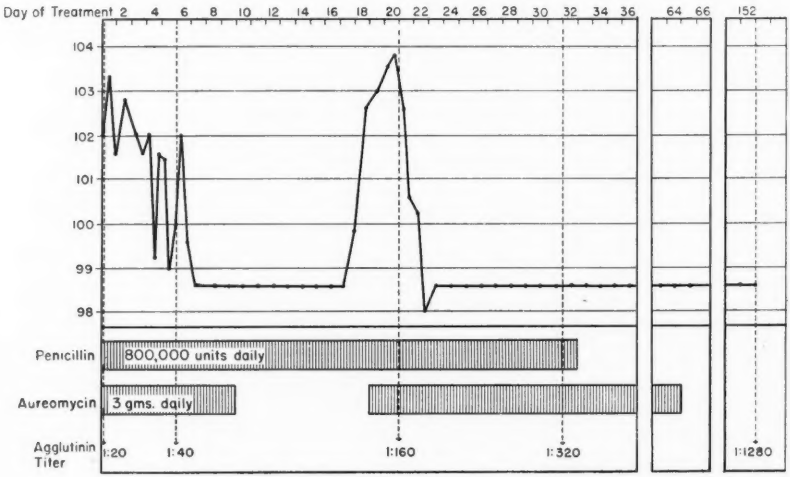


Fig. 3.—Temperature graph showing medication and agglutination titers.

Course in the Hospital.—Bedside chest film in the supine position confirmed the clinical impression of pathology in the right hemithorax (Fig. 2, *B;* Fig. 2, *A* is a thoracic roentgenogram taken during previous admission). A lumbar puncture produced an initial pressure of 16 cm. Initial treatment included parenteral fluids and penicillin (500,000 units immediately and 100,000 units every three hours), and Aureomycin in doses of 0.5 Gm. every four hours for eight days. Nuchal rigidity disappeared during the first four days, coincident with defervescence and clearing of the sensorium. Eight days later, high fever, delirium, nuchal rigidity, and a headache recurred. A lumbar puncture at the time

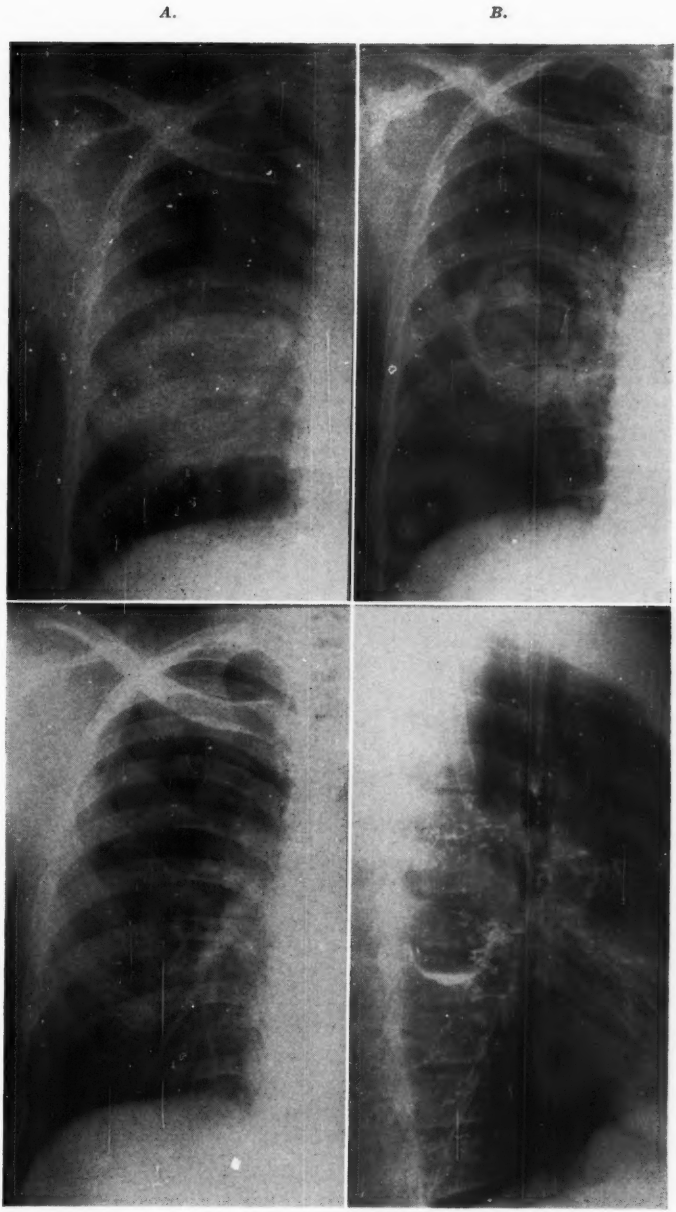


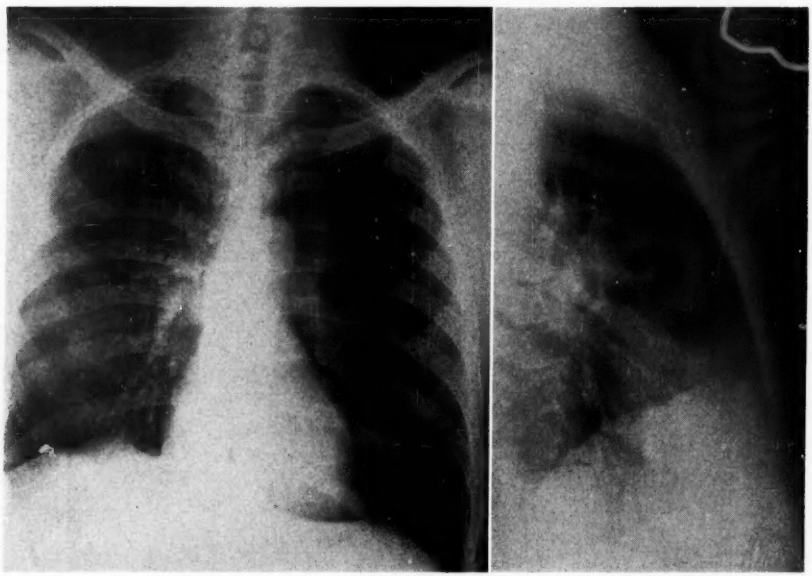
Fig. 4.—*A*, Homogeneous density of apical portion of the right lower lobe is seen in roentgenogram of Sept. 1, 1950.

B, Cavitation is fully developed in the consolidation of the right lower lobe four weeks after admission, Sept. 21, 1950.

C, Twelve weeks, (Nov. 16, 1950) after admission, a thin-walled cavity is seen in the superior segment of the right lower lobe.

D, Bronchogram of Nov. 30, 1950, demonstrating communication of tracheobronchial tree with residual cavity. Fluid level evident.

revealed purulent spinal fluid. After four days of Aureomycin therapy, clinical improvement was noted. The Aureomycin was continued for six weeks in doses of 3.0 Gm. daily (Fig. 3). The jaundice gradually disappeared and the liver function tests were normal during the fifth week. A roentgenogram made on September 1, in the upright position, revealed a discrete homogeneous density involving the apical portion of the lower lobe (Fig. 4, *A*). A bronchoscopy, on September 18, demonstrated acute and chronic tracheobronchitis with a small amount of secretion coming from the bronchus of the superior segment of the lower lobe. Central rarefaction of the lesion was observed on a roentgenogram taken September 11, and further cavitation was noted on September 21 (Fig. 4, *B*). The patient gained weight and strength with his afebrile course. Despite the pulmonary pathology, only a mild cough with minimal expectoration of grayish-black mucoid sputum persisted. Studies for *Pasteurella tularensis* were not carried out on the sputum. After ambulatory status was obtained by the patient, a follow-up film, on November 16, demonstrated a 5 by 3 cm. thin-walled cavity without any surrounding infiltration (Fig. 4, *C*). Subsequently, a bronchogram demonstrated communication with the tracheobronchial tree (Fig. 4, *D*). There was no evidence of bronchiectasis of the surrounding parenchyma.



A. *B.*

Fig. 5.—*A* and *B*. Roentgenograms taken on March 12, 1951, one month after discharge from the hospital. Diaphragm is higher on the right than previously.

On December 2, the Medical-Surgical Chest Conference recommended resection of the residual cavity which communicated with the bronchus intermedius and had remained stationary in size for several weeks. The patient agreed to surgery and was transferred to the Thoracic Surgical Service. Sputum examinations revealed *B. proteus* on culture. Streptomycin was therefore given, as well as aerosol and parenteral penicillin. The former drug was given also because the tularemic infection had been of the typhoidal type. A thoracotomy was performed on Jan. 5, 1951. Adhesions from the parietal pleura to the superior segment of the right lower lobe were severed. A cavitary lesion could then be palpated in the superior segment which was subsequently resected. Postoperatively, the

patient's course was uneventful, except for the development of an air pocket after the removal of the drainage tubes. This was corrected by continuous suction through an intercostal catheter. He was discharged on Feb. 6, 1951. Follow-up examination one month later revealed no significant physical or roentgenologic abnormalities (Fig. 5, *A*, *B*).

Pathologic Report.—Doctors O. J. Wollenman and B. E. McCain reported the presence of a cavity measuring 7 by 3 cm. which communicated with the lower lobe bronchus and one of its branches. Grossly, the inner surface of the cavity was trabeculated, smooth, and glistening. Its wall measured 1 to 2 mm. in thickness (Fig. 6). Microscopically, the lining was that of respiratory epithelium. The wall was composed of fibrocollagenous tissue in which there were rare lymphocytes and plasma cells. Nodular collections of lymphocytes, containing also occasional small giant cells with 4 to 5 centrally placed nuclei and eosinophilic cytoplasm, were seen peripherally. Interstitial fibrosis replaced the normal architecture of the adjacent lung. The presence of respiratory epithelium and lack of carbon pigment in the cavity wall suggested a cyst of congenital origin. However, the fact that the wall was made of fibrocollagenous tissue supported the thesis that it was an acquired cystic lesion.

Fig. 6.—Residual cavity of the superior segment of the lower lobe of the right lung is seen in the pathologic specimen. The bronchial communication is indicated with a match stick.

DISCUSSION

The mortality rate of tularemic pleuropulmonary disease has been significantly altered by modern antibiotic therapy. Analyzing 109 cases of pulmonary tularemia occurring before the advent of streptomycin, Francis[1] found the death rate to be 46.7 per cent. Four years later, in 1945, Stuart and

Pullen[2] reported a mortality rate of 39.9 per cent in 268 cases. These figures illustrate the relatively high death rate associated with pulmonary involvement in contrast to that of 7.4 per cent from all other forms of tularemia.[3] Since the demonstration of the bactericidal action of streptomycin on *Past. tularensis* by Heilman in 1944,[4] there has been a sharp decline in fatalities. This is shown in the combined series of Hunt[5] and Morgan.[6] Of 27 patients with tularemic pneumonia treated with streptomycin, only one died. This death was due to pulmonary embolism during convalescence.

The only known patient who survived a tularemic lung abscess,[7] in addition to those reported above, was given chloramphenicol and streptomycin with complete disappearance of the pulmonary lesion. As laboratory studies are of little value in early diagnosis of tularemia, Francis[3] and others have advocated the use of streptomycin as a presumptive diagnostic test in cases of pneumonia of undetermined etiology. Morgan[6] and Hunt[5] emphasized that 16 of their 27 patients presented themselves with the sole finding of an obscure bronchopneumonia, many of which were considered viral in origin. The immediate use of streptomycin in patients seriously ill with pneumonia was apparently lifesaving in several instances.

Aureomycin has been found to be equally effective.[8, 9] This antibiotic has the obvious advantages of a broad spectrum activity, effectiveness by oral administration, and a lessened incidence of toxicity. The loss of specificity in the diagnostic test when Aureomycin is substituted for streptomycin would seem unimportant, particularly since neither agent inhibits diagnostic rise of titer of specific agglutinins. Our second patient was treated initially with the presumptive diagnosis of leptospirosis, the correct diagnosis of tularemia being made only after the rise in agglutinin titers to diagnostic levels. The need for prolonged therapy in the patient ill with tularemia is illustrated by the same case. The first patient was suspected of having tularemia because of the history of exposure to rabbits and squirrels. He responded to Aureomycin and had a rapid increase in titer for tularemia.

PATHOLOGY

The pleuropulmonary complications of tularemia have become recognized in greater numbers since Verbrycke[10] published the first autopsy of the typhoidal type of the disease in 1924. According to Archer and associates,[11] 90 per cent of the patients with the disease have abnormal thoracic roentgenograms.

Tularemic pleuropulmonary disease is important for it simulates the pathology of pulmonary tuberculosis, namely, nodular bronchopneumonia, lobar consolidation, varying degrees of tracheobronchitis, and pleurisy with effusion. These lesions are considered integral parts of the disease and not due to secondary bacterial invasion. Although very few cases have been described, an additional pathologic lesion of the lung in tularemia is that of abscess. Blackford[12] reported the first case in 1932 (Table I). Many other writers, according the Blackford, have remarked on the great similarity between the microscopic

pictures seen in the tubercle of tuberculosis and in the caseous necrosis of tularemia.

Occasionally cavitation is mentioned in reports of tularemia involving the lungs and pleura. Lillie and Francis[13] have carefully tabulated such data up to 1936. Since necrosis is a basic pathologic process of tularemia, focal necrosis has been observed frequently at post-mortem examinations of the patients dying from the disease. Case 3 (Table I) was reported by Moss and Weilbaecher[14] as showing multiple small areas of focal necrosis scattered throughout areas of consolidation. However, Pullen and Stuart[15] reported the autopsy to demonstrate multiple lung abscesses. Kavanaugh[16] mentioned one case of pneumothorax presumably secondary to a focal abscess.

In a clinical study of 600 patients with tularemia, Foshay[17] found the incidence of pneumonia varied from 9.1 per cent in the oculoglandular form to 52.2 per cent in the typhoidal type, with an over-all incidence of 18 per cent. One lung abscess was reported in this series but not in any detail.

TABLE I

CASE NUMBER	AGE	SEX	RACE	SOURCE	DIAGNOSED BY	LOCATION	AUTHOR
1	38	M	C	Opossum	Autopsy	RLL	Blackford
2	22	M	C	No history	Autopsy	Left lung	Moss and Weilbaecher
3	21	F	C	Unknown	Autopsy	Multiple	Moss and Weilbaecher Pullen and Stuart
4	29	M	W	Unknown	Agglutination tests	LUL	Ray and Warren
5	36	M	C	Rabbits and squirrels	Agglutination tests	RLL	Case 1, herein
6	44	M	C	Unknown	Agglutination tests	RLL	Case 2, herein

ABSCESS VERSUS CYST

From the pathologist's report of our second case, one might conclude that a pre-existing cyst was excised. However, the patient's thoracic roentgenogram taken a year earlier demonstrated no evidence of a cyst in the right lower lobe. Such a conclusion also fails to recognize the basic pathologic process in tularemia, the pathogenesis of advanced lesions of the disease, and the clinical course of the patient. Cavitation in the tularemic pneumonia resulted from the coalescing of focal necrosis with absorption and expectoration of the central necrotic debris. Larger amounts of sputum were expectorated during the time cavitation was developing. Thus, internal drainage, via the communications with the tracheobronchial tree, was performed with the aid of the patient's coughing. The bronchial communications were also the source of the epithelium which lined the abscess cavity (see pathologist's report). Sinus tracts of externally (surgical) drained pulmonary abscesses become lined by the same type of epithelium as that of the skin. It is for this reason they sometimes remain open, do not heal primarily, and require excision. The failure of the cavity in this case to heal was due to the epithelial lining and absence of external pressure. The cavity failed to decrease in size when the process of epithelization was completed.

Excision of the superior segment of the lower lobe of the right lung was done in the second case as an elective procedure after recovery from the generalized infection of the typhoidal type. In retrospect, early drainage might have resulted in a collapse of the abscess cavity and prevented the necessity for subsequent thoracotomy. The patient was very ill at the time cavitation of the pulmonary lesion was evident. Rapid improvement after proper therapy dictated continuance of medical management to maximal benefit.

SUMMARY AND CONCLUSIONS

1. Two additional cases of tularemic lung abscesses in which the patients survived, due to antibiotic therapy, are reported. Only 4 other cases of this condition have previously been described in any detail. Meningitis and hepatitis were also present in one of the 2 patients.

2. The difficulties of early diagnosis and the use of the presumptive test with antibiotics are cited. The diagnoses in the cases herein reported were established by a delayed rise in agglutination titers for tularemia.

3. The need for the thoracic surgeon to know of the pleuropulmonary complications of tularemia is demonstrated.

4. The first report of segmental pulmonary resection in tularemia is presented.

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BENIGN GRANULAR-CELL MYOBLASTOMA OF THE BRONCHUS: REPORT OF TWO CASES

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THE tumor known as benign granular-cell myoblastoma is rarely found associated with the respiratory tract. In 1949, Murphy, Dockerty, and Broders[13] listed 12 cases in which myoblastoma was associated with the respiratory tract (in one with a bronchus) in their review of 229 cases of benign and malignant myoblastomas from the world literature. Bissi and Magri[3] recorded the finding of 18 of these tumors in respiratory tracts in a recent review of 378 examples of benign and malignant myoblastomas of known location. Stout[18] reported 5 cases in which the larynx or trachea was affected, in a series of 120 examples of benign myoblastomas seen in the surgical pathology laboratory of Columbia University. We have reviewed the literature pertaining to involvement of the bronchi by this entity and were able to find only 5 previously reported cases.[8-11, 16] The purpose of this communication is to describe 2 cases of benign granular-cell myoblastomas of the bronchi that were recently encountered at the Mayo Clinic.

A granular-cell myoblastoma may be defined as a distinctive, benign tumor that is composed of large, polygonal cells, with small, dark nuclei and conspicuous, acidophilic, cytoplasmic granules. Since the identification of this tumor as a specific oncologic entity by Abrikossoff, in 1926,[1] confusion and conjecture pertaining to its derivation, composition, and classification have been manifest in the literature. In 1931, Abrikossoff[1] published additional data pertaining to this entity and described four different varieties. The first type would appear to correspond to what today is usually considered to represent a benign granular-cell myoblastoma, while the remaining three types have been interpreted in a varied and confusing manner by various authors in subsequent articles.[7]

Stout[18] has resolved the problem to some extent by recognizing a benign and malignant type of myoblastoma. The benign variety (usually designated as "granular cell myoblastoma," or "uniform myoblastoma") corresponds to the Type I tumor described by Abrikossoff, while the malignant variety is composed of two subtypes; the first, and rarer type, is distinguished primarily by the pleomorphism of the nuclei and the second type is singular because of its

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organoid pattern, plus varying degrees of cellular pleomorphism. In this manner, Stout has separated the malignant myoblastoma from the rhabdomyosarcoma, a point which in the past has caused much confusion and overlapping in the diagnosis of the two entities. To complicate further the understanding of the malignant myoblastoma (also known as pleomorphic myoblastoma), there are those who refuse to accept its relationship to the benign form and prefer to consider it as representing a malignant nonchromaffin paraganglioma,[17] an alveolar sarcoma of soft parts,[4] or a rhabdomyosarcoma.[7]

Because the nature of the benign and malignant varieties is obscure and because the varied sites of origin are often devoid of striate muscle, a host of opinions have developed concerning its progenitor. The early reports in the literature considered this tumor to be a neoplasm and designated the tumor as a myoblastoma because of the similarity of the cell type involved to the proliferating embryonal myoblast. Today there are two main schools of thought concerning histogenesis. The first school considers that this is a neoplasm and that it is derived from myoblasts, a concept adhered to by Stout[18] and supported by the tissue culture studies of Murray.[14] Fust and Custer,[6] on the other hand, have considered the benign variety to be a neoplasm of nerve origin and have proposed the term "granular cell neurofibroma." Those neoplasms arising in striate muscle, according to these authors, should be considered to be either rhabdomyomas, if benign, or rhabdomyosarcomas, if malignant. The second school disbelieves that they are neoplasms and considers only the benign variety in discussing the problem. Histiocytes, altered fibroblasts,[15] and degenerative or regenerative changes in striate muscle have all been proposed as responsible for the distinctive cell of this tumor.[2] It is not the purpose of this report to support or decry any of these theories of origin, but rather to describe a specific tumor as defined in the second paragraph, an entity that is generally identified as a benign granular-cell myoblastoma.

The granular-cell myoblastoma, benign or malignant, is a relatively uncommon tumor as evidenced by the reviews of the literature by Murphy, Dockerty, and Broders[13] and more recently by Bissi and Magri.[3] The latter authors described 13 personal cases and were able to collect 439 cases from the world literature including 36 cases of congenital epulis (an entity that is not accepted by all authors as being related to the granular-cell myoblastoma).[5] The incidence of malignancy is difficult to establish, but is probably low. According to Stout's[18] series, it is about 12 per cent. Without doubt, in many of the reported cases, the malignant variety in the deep soft parts is probably better considered as rhabdomyosarcoma. No examples of the malignant variety have been reported in the bronchi. While the granular-cell myoblastoma may be found in almost any tissue of the body with the possible exception of the central nervous system,[12] more than 50 per cent of them have arisen in the tongue, skin, and subcutis. The age of the patients has ranged from the first through the ninth decade with a peak incidence in the fourth and fifth decades. The gross appearance of the benign myoblastoma is not distinctive. Usually it appears as a small, well-circumscribed nodule or an indurated area, and when associated with a mucous membrane, it may be pedunculated. It is rare for the benign

variety to exceed 2 cm. in diameter. Because of their small size, lack of a capsule, and firm consistency, they have been mistaken for scirrhous carcinomas when found in association with breast tissue.

REPORT OF CASES

CASE 1.—This 41-year-old white man was admitted to the hospital in May, 1953, because of hemoptysis, chills, fever, and pain of two days' duration in the anterior part of the thorax. He had been bothered with intermittent episodes of asthmatic bronchitis since 1937 and also had progressive muscular dystrophy. The latter had been diagnosed thirteen years before admission. Physical examination showed the patient to be a thin, asthenic male who appeared to be chronically ill. He coughed intermittently and had a temperature of 100.3° F. Muscular atrophy over the shoulder girdles was noted. On auscultation, râles, rhonchi, and expiratory wheezes were heard over both lung fields but were more pronounced on the left. The edge of the liver was palpable 4 fingerbreadths below the right costal margin.

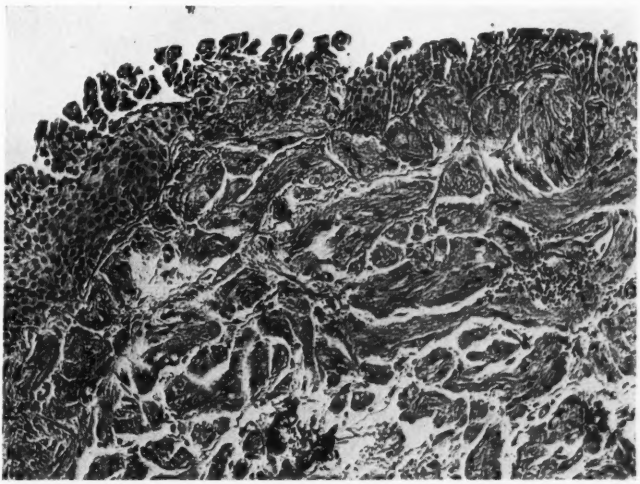

Fig. 1.—(Case 1). Biopsy specimen featuring syncytiumlike mass and polygonal cells that exhibit acidophilic granules in cytoplasm. Foci of squamous metaplasia are shown in the overlying respiratory epithelium (hematoxylin and eosin; ×140).

Routine laboratory work was essentially normal. The roentgenogram of the thorax showed only thoracic scoliosis. Smears and cultures of the sputum were negative for fungi, tubercle bacilli, and malignant cells. Both skin tests for tuberculosis with first and second strength purified protein derivative tuberculin were negative. Bronchoscopic examination revealed a ridge in the right upper lobe bronchus that obscured, but did not occlude, the orifice to the anterior segmental bronchus. The surrounding bronchial mucosa was red and appeared chronically inflamed, but no source of bleeding was evident. Tissue removed for biopsy from the ridgelike area proved to be a granular-cell myoblastoma and the remainder of the tumor was removed by endobronchial manipulation one week later.

The patient had another episode of hemoptysis in January, 1955. At that time, smears of the sputum were negative for acid-fast bacilli and malignant cells. Roentgenograms of the thorax failed to disclose any evidence of tumor or secondary obstructive pneumonitis. Bronchoscopic examination was thought inadvisable because of the patient's poor pulmonary status secondary to chronic asthmatic bronchitis and progressive muscular dystrophy.

Pathologic Changes.—The material received from the endoscopic removal of the ridge-like thickening in the right upper lobe bronchus consisted of several nondescript pieces of tissue that aggregated approximately 6 mm. in diameter. The microscopic sections revealed fascicles and strands of cells with and without distinct cell boundaries that contained fine, acidophilic, cytoplasmic granules and small, dark nuclei. No mitotic figures were seen. These cells frequently formed syncytiumlike masses that were interposed between strands of collagen. The deeper portions of the tumor demonstrated interdigitation with a few strands of smooth muscle and an occasional small nerve filament. The overlying mucous membrane was intact and separated from the cells of the tumor by an intact basement membrane. Foci of squamous metaplasia were noted in the overlying respiratory epithelium. There was a minimal amount of small round-cell infiltration at the periphery of the tumor. No cartilage was demonstrated in the sections (Fig. 1).

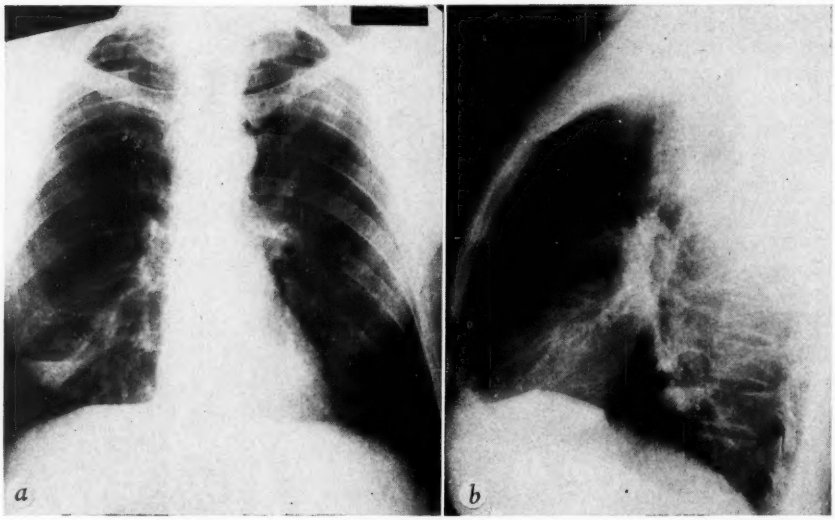

Fig. 2.—(Case 2). Fluid level in thin-walled cyst. The area of increased density in the superior medial aspect of the cyst was considered to represent anomalous bronchi or blood vessels. *a*, Anteroposterior view. *b*, Lateral view.

Case 2.—A 44-year-old white man, a tool-grinder, was referred to the Clinic in October, 1955, because of findings in roentgenograms of the thorax, taken six months and one month prior to registration, that were consistent with a diagnosis of pulmonary cyst in the right lower lobe of the lungs. The patient had originally consulted the Clinic thirteen years before because of pain in the left upper part of the chest. At that time, physical examination and roentgenograms of the thorax were negative, and the pain was considered muscular in origin. The patient had sought medical advice eighteen months prior to his last registration because of weakness, drowsiness, and a run-down feeling. His only respiratory complaint was of a chronic cough productive of a minimal amount of mucoid sputum.

Physical examination at the Clinic in October, 1955, gave essentially negative results.

Routine laboratory tests were within normal limits. Smears of the sputum revealed no acid-fast bacilli. Skin tests with first strength P.P.D. tuberculin were negative, while those with the second strength P.P.D. tuberculin were positive. Roentgenograms of the thorax were interpreted as showing the presence of a partly fluid-filled, thin-walled cyst in the posterior basal segment of the right lower lobe. There was no evidence of inflammation

in the surrounding parenchyma of the lung. At the superior medial aspect of this cyst a shadow of increased density, 1.5 cm. in diameter, was considered to represent anomalous bronchi or blood vessels in association with the cyst (Fig. 2, *a* and *b*). Bronchoscopic examination was performed but revealed nothing of note.

Operation was advised, but delay was necessary because of an upper respiratory infection. On Jan. 26, 1956, a segmental resection of the basilar segments of the right lower lobe was performed. The patient's postoperative course was uneventful.

Pathology.—The specimen consisted of the basilar segment of the right lower lobe of the lung. Beginning 8 mm. above the origin of the posterior basilar segmental bronchus was a sessile, polypoid tumor which occluded approximately 80 per cent of the bronchial lumen.

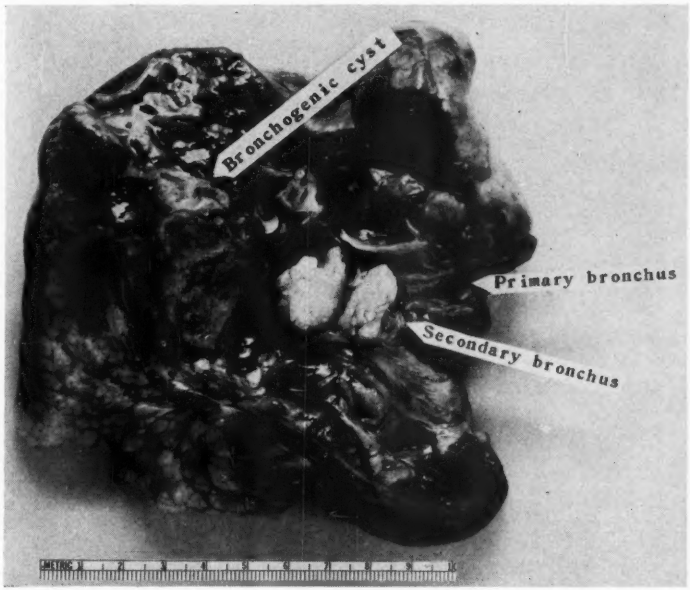

Fig. 3.—(Case 2). Posterior basilar segment of right lower lobe of lung demonstrating the relation of the tumor to the bronchi and cyst. The slightly lobulated, polypoid nature of the tumor is shown. The bronchogenic cyst has been bisected.

The exposed surface of the tumor was slightly lobulated, rough and dull. On cross section the tumor was found to involve approximately 60 per cent of the circumference of the bronchus and to measure 2 cm. in diameter. The bronchial wall had been infiltrated, and the peribronchial tissues were involved to a depth of one centimeter. The advancing edge of the tumor was well circumscribed, and two small peribronchial lymph nodes were compressed but not infiltrated by the mass. The cut surface was a nondescript grayish-white with suggested bundles and whorl formation. Five millimeters distal to the mass, the posterior basilar segmental bronchus communicated freely with a spherical thin-walled cyst that measured 6 cm. in diameter. The cyst wall was uniform and measured 2 mm. in thickness. The lumen was partially filled with white mucoid secretions (Fig. 3).

Microscopic sections of the tumor revealed moderately large cells that contained conspicuous acidophilic cytoplasmic granules (Fig. 4). Many of these granules stained red with periodic acid-Schiff reagent and black, with Sudan black B. For the most part, the nuclei were uniform in size and contained a delicate chromatin network. The nucleoli were of moderate size and eccentrically placed. No mitotic figures were seen. In the

polypoid portion of the tumor, these cells formed sheets with rather sparse interspersion of collagen fibers, while in the deeper portions they were arranged in strands and bundles between more conspicuous collagen fibers. The bronchial mucous glands and cartilage rings were surrounded by the tumor without evidence of infiltration. In the deeper reaches of

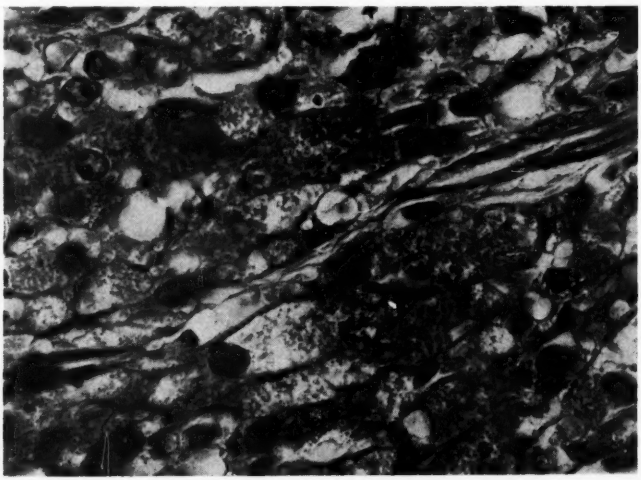

Fig. 4.—(Case 2). The granular nature of the cell cytoplasm and the benign nature of the nuclei are well demonstrated (Masson's trichrome; ×800).

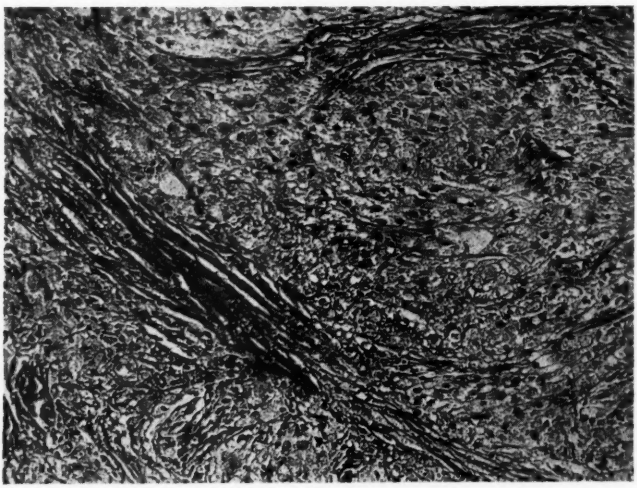

Fig. 5.—(Case 2). Deep segment of tumor demonstrating the relationship of the tumor cells to the bundles of collagen (Masson's trichrome; ×150).

the tumor, the connective tissue was more prominent and there was no evidence of encapsulation (Fig. 5). Smooth muscle was not found in association with the tumor cells. Several small nerves were observed at the infiltrating edge of the tumor. The overlying respiratory epithelium had undergone marked squamous metaplasia with conspicuous keratinization on the surface (Fig. 6). Pseudoepitheliomatous hyperplasia was not a conspicuous feature except in tissue sections cut in a tangential plane to the surface of the tumor.

Microscopic examination of sections taken through the cyst wall revealed a bronchogenic cyst that was lined throughout by a columnar ciliated respiratory type of epithelium. The wall of the cyst was composed primarily of bundles of collagen interspersed with smooth muscle fibers and occasional small mucous glands. No cartilage was demonstrated in the

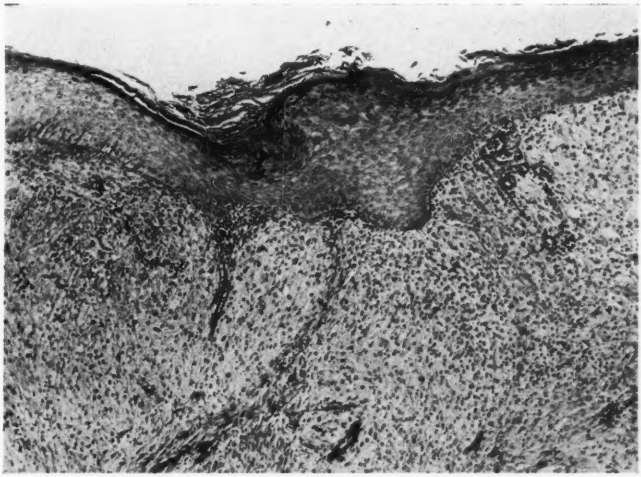

Fig. 6.—(Case 2). Epidermization and keratinization of the respiratory epithelium on the surface of the tumor are pronounced. Sheets of granular cells abut intact basement membrane (Masson's trichrome; ×70).

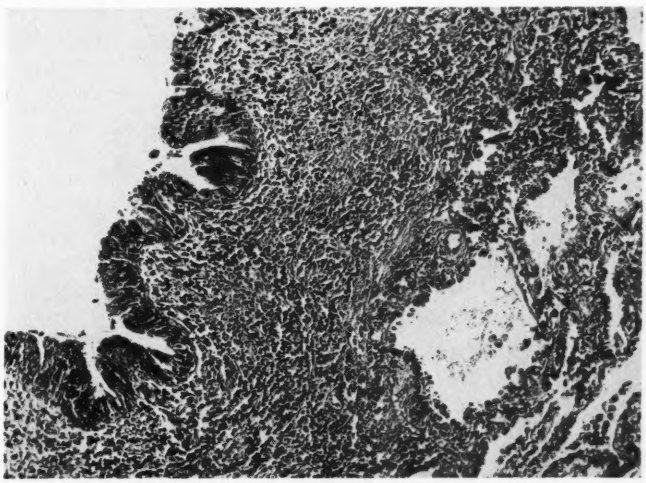

Fig. 7.—(Case 2). Representative section through bronchogenic cyst revealing intact respiratory epithelium, thin fibrous wall, and relative paucity of inflammatory cells (hematoxylin and eosin; ×100).

cyst wall. A moderate number of lymphocytes and a few plasma cells were scattered throughout the wall of the cyst (Fig. 7). In the surrounding parenchyma of the lung, some thickening of the alveolar septa and prominent alveolar lining cells were demonstrated,

TABLE I. PERTINENT DATA IN FIVE CASES REPORTED IN LITERATURE AND OUR TWO CASES

AUTHOR	AGE, RACE, AND SEX	LOCATION	GROSS CHARACTER AND SIZE	CLINICAL SYMPTOMS	TREATMENT	FOLLOW-UP
Kramer 1939	15 F	R.L.L. bronchus	Sessile, polypoid; size (?)	Recurrent obstructive pneumonitis	Endoscopic removal plus coagulation	5 months; x-rays negative
Kraus, Melnick, and Weinberg 1948	48 M	Right-main bronchus	Filled lumen of bronchus; infiltration of bronchus, 6 by 5 by 3.5 cm.	Recurrent pneumonitis; empyema with bronchopleural fistula; 6-year history	Pneumonectomy; prior biopsy	Died after operation
Lowbeer 1953	24 F*	Left-main bronchus	Polypoid; no infiltration of bronchus; size (?)	Wheezing; productive cough; atelectasis	Pneumonectomy; prior biopsy	?
Liebow 1952	59 M*	L.U.L. bronchus	Polypoid 3 by 4 by 5 mm.; squamous metaplasia	?	?	?
Ramsey 1955	18 F	Right-main bronchus	Lobulated firm mass, 2 cm. with partial bronchial obstruction	Recurrent pneumonia	Endoscopic removal	4 weeks; negative bronchoscopic examination
Authors Case 1	41 M	R.U.L. bronchus	Ridgelike mass, squamous metaplasia; aggregate 6 mm. in diameter	Chronic asthmatic bronchitis; hemoptysis	Endoscopic removal	2 years; no evidence of obstruction in x-rays
Authors Case 2	48 M	R.L.L. posterior basal segmental bronchus	Sessile, polypoid 2 cm. squamous-cell metaplasia and leukoplakia; infiltration of bronchus	Chronic slight productive cough; bronchogenic cyst by x-ray	Segmental resection	Recent

*Patients were Negroes.

but there was no evidence of pneumonitis, ancient or recent. The remaining portion of the parenchyma of the lung was essentially normal. The peribronchial lymph nodes were not remarkable.

The pertinent data in the 5 cases reported in the literature and in the 2 cases presented in this report are summarized in Table I.

COMMENT

From the clinical standpoint, the symptomatology appears to be related to obstruction of a bronchus with varying degrees of obstructive pneumonitis. The degree of obstruction was of sufficient magnitude in 4 of the 7 cases recorded in Table I to warrant bronchoscopy on this basis alone. In our Case 2, the patient was asymptomatic and underwent operation primarily for a bronchogenic cyst. The nodular density visible in the roentgenograms in close proximity to the superior aspect of the cyst was considered to represent anomalous bronchi or blood vessels. Bronchoscopy in this case gave negative results. The high degree of obstruction as demonstrated in the surgical specimen probably played a role in the retention of the secretions in the bronchogenic cyst and at a later date might have produced infection of the bronchogenic cyst and surrounding parenchyma.

In our Case 1, the myoblastoma was an incidental finding at bronchoscopy. This patient had asthma, and bronchoscopic examination was performed only as a means of investigating the history of hemoptysis. In all probability, this small tumor contributed little or nothing to the symptoms of this man.

The location of these tumors may be summarized as follows: involvement of the main bronchus, three times (two right, one left); lobar bronchi, three times (right lower lobe, left upper lobe, right upper lobe), and segmental bronchus, one time (right lower lobe posterior).

The gross character of the tumors was rather uniform except in the case described by Kraus, Melnick, and Weinberg[9] and in our Case 1. The majority of the tumors may be described as sessile, polypoid tumors varying from 0.5 to 2 cm. in diameter. Infiltration into the wall of the bronchus was demonstrated in two of the three surgical specimens removed by pneumonectomy or segmental resection. Endoscopic removal in 3 cases precludes any exact statement as to the degree of infiltration into the bronchial wall. The mode of therapy in Liebow's[10] case was not disclosed.

Squamous-cell metaplasia was found in 3 of the cases. Liebow[10] commented on the pseudoepitheliomatous hyperplasia in his specimen and mentioned the common occurrence of this phenomenon in relation to these tumors in the tongue and skin.

It is obviously impossible to make any rigid statements about treatment on the basis of these few cases. However, we consider that the management is akin to that of bronchial adenoma. It seems reasonable to recommend a conservative surgical approach. A most important modifying feature would be the amount of obstructive pneumonitis and secondary suppurative disease. Endoscopic manipulation can play an important role in maintaining an airway, particularly in the patients whose poor general condition contraindicates

operation but the extrabronchial invasion of the tumor places some limitations on complete endoscopic control. The low incidence of malignancy seems to justify the most conservative operative management. However, careful follow-up supervision is necessary because the neoplasm may recur if removal is not complete.

SUMMARY

Two cases of asymptomatic benign granular-cell myoblastoma of the bronchus encountered at the Mayo Clinic and 5 cases previously reported in the literature form the basis of this report. No example of a malignant myoblastoma of the bronchus was found in the literature. The tumor presents most often as a polypoid sessile mass and causes varying degrees of bronchial obstruction. Obstructive pneumonitis appears to be the most usual clinical and roentgenologic finding. Pseudoepitheliomatous hyperplasia may be observed in the epithelium overlying the tumor and should not be confused with squamous-cell epithelioma. Infiltration through the bronchial wall was observed in 2 of 3 cases in which specimens of lung were available for examination. Although endoscopic removal of the tumor would appear feasible under certain circumstances, careful follow-up of the patient is necessary as this neoplasm may recur if incompletely excised. Malignant change superimposed on the benign granular-cell myoblastoma has not been recorded.

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THE PROPHYLAXIS OF CARDIAC ARRHYTHMIAS COMPLICATING PULMONARY SURGERY

A Preliminary Report

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In 1943, Bailey and Currens simultaneously published the first reports of cardiac arrhythmias complicating the early postoperative course of thoracic surgery.[1, 2] In the ensuing years, although the occurrence of this type of surgery has multiplied, reports of this arrhythmic complication have been few and infrequent.[3-5]

At the Henry Ford Hospital, it seemed that this complication occurred rather frequently and the paucity of reports was not due to the disappearance of the syndrome. A two-year review of pneumonectomies and lobectomies was therefore made with special attention to the pre- and postoperative cardiac status.

MATERIAL

During the years 1953 and 1954, thirty-six pneumonectomies and forty-three lobectomies were performed. Of these, 34 and 42 patients, respectively, survived the surgery and comprise the study. Ten of the patients who had had pneumonectomies and 6 with lobectomies had postoperative arrhythmias and the case summaries are presented.

Case 1.—A 55-year-old man had an epidermoid carcinoma of the right bronchus. The heart was normal prior to operation. On the third day following right pneumonectomy, he developed a rapid atrial fibrillation. He was given intravenous Cedilanid. The next day the heart rhythm reverted to sinus tachycardia and the patient developed evidence of atelectasis or pneumonia. A diarrhea began after two doses of Aureomycin. Paralytic ileus was relieved by Prostigmin but diarrhea recurred followed by cardiac failure, pulmonary edema, and death. Autopsy did not disclose any evident cardiac abnormality. There was marked gastroenteritis, not of the pseudomembranous type.

Case 2.—A 51-year-old white woman, who had had a rectal resection seven years previously for adenocarcinoma, was admitted with a right lung metastasis. The heart seemed normal prior to surgery. On the fifth day following the right pneumonectomy, it developed a rapid arrhythmia which proved to be multifocal atrial ectopic beats. Quinidine increased the rate but the ectopic beats continued. Digitalization slowed the rate but did not abolish the ectopic rhythm. On the thirteenth postoperative day, the heart became regular and remained so.

Case 3.—A white man, aged 62, had an adenocarcinoma of the right lung and a normal heart preoperatively. The second day following right pneumonectomy, his heart developed a rapid atrial fibrillation which slowed with digitoxin and was converted to normal rhythm on

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the fourth postoperative day with quinidine. The heart then alternated between nodal tachycardia and sinus rhythm until the seventh postoperative day when it was slow and regular. This continued except for a few ectopic beats.

CASE 4.—A white man, aged 56, with an undifferentiated squamous-cell carcinoma of the right lung and a normal preoperative heart, developed atrial fibrillation on the second day after a right pneumonectomy. He was given 0.8 mg. of Cedilanid and the heart converted to sinus rhythm later that day with no further trouble.

CASE 5.—A white man, aged 59, with adenocarcinoma of the right lung had a normal heart clinically. It began rapid atrial fibrillation on the second postoperative day following a right pneumonectomy. The patient was given 0.8 mg. and 0.4 mg. of Cedilanid and the following day developed sinus tachycardia. This continued over the next three days with occasional runs of atrial fibrillation. The heart became completely regular again with quinidine and remained so.

CASE 6.—A white man, aged 50, had carcinoma of the lung and no evident preoperative cardiac disease. On the third day following right pneumonectomy, he developed a rapid atrial fibrillation. He was digitalized with Cedilanid, receiving a total of 3.4 mg. in thirty hours plus 0.4 mg. of digitoxin. The rate was not controlled and he went into ventricular failure and shock. Multiple ventricular ectopic beats became evident and he died on the fifth postoperative day. Necropsy revealed slight herniation of the heart into the mediastinum. The heart was dilated and weighed 450 Gm. There was slight thickening of the posterior leaflet of the mitral valve. Marked subendocardial hemorrhage was found, along with marked stenosis of the right and left coronary arteries, without occlusion.

CASE 7.—A 60-year-old white man had epidermoid carcinoma of the lung and no evident heart disease. On the day following a left pneumonectomy, the pulse was irregular but no electrocardiogram was made. The following day, without treatment, there was a sinus tachycardia.

CASE 8.—A 60-year-old white man had a four-year history of recurrent paroxysmal atrial fibrillation, precipitated by a nodular goiter, which continued after removal of the goiter. He had a left pneumonectomy for carcinoma of the lung and had an episode of atrial fibrillation both the day before and the day after surgery. Quinidine was effective in converting both episodes to sinus rhythm.

CASE 9.—A white man, 54 years old, had an epidermoid carcinoma of the lung and no evident cardiac disease, preoperatively. On the fifth day after a left pneumonectomy, he developed an atrial fibrillation. He received 0.8 mg. of Cedilanid with continuation of the fibrillation. On the seventh day he had 0.4 mg. of Cedilanid and that day the heart returned to regular rhythm. A right bundle branch block persisted after return to normal rhythm.

CASE 10.—A white man, aged 58, with adenocarcinoma of the lung and thyroid, had no immediate complication following left pneumonectomy but returned on the thirtieth postoperative day with hemothorax, abdominal metastases, and a tachycardia with many ectopic auricular beats. These continued occasionally until death, eleven days later. At necropsy, the heart was displaced to the right with dilatation of the right atria and ventricle and 75 c.c. of yellow-orange fluid in the pericardial sac. The coronary arteries were normal. The heart weighed 500 Gm.

CASE 11.—A white woman, aged 37, with tuberculosis, had a normal heart preoperatively. On the sixth day following a left upper lobe resection, she developed a supraventricular tachycardia which converted after 0.4 mg. of Cedilanid.

CASE 12.—A 64-year-old white man had an epidermoid carcinoma and a right upper lobectomy. On the third postoperative day, he had numerous extrasystoles. On the tenth

postoperative day, he developed atelectasis of the right lung with empyema and had numerous atrial ectopic beats. On the thirteenth postoperative day, the rhythm was very irregular with runs of multifocal atrial ectopic beats. Digitalis was given and the heart became normal again with only occasional ectopic beats.

CASE 13.—A white man, aged 61, with adenocarcinoma of the lung had a normal heart preoperatively. He had a right lower lobectomy and, during surgery, it was noted that he became cyanotic if the right lung was deflated. He did well in the immediate postoperative period but was readmitted on the twenty-first postoperative day with empyema, and a rapid atrial fibrillation which converted after 1.2 mg. of Cedilanid. The empyema was drained and he had no further complications.

CASE 14.—A white man, aged 60, had an adenocarcinoma of the lung and a normal heart. On the fourth day following a right lower lobectomy he developed atrial fibrillation. He was digitalized and the heart reverted to normal rhythm and digitalis was continued. On the sixth postoperative day, the heart again fibrillated with spontaneous conversion. This was repeated on the twelfth postoperative day. Digitalis was discontinued and quinidine instituted and no further arrhythmias occurred.

CASE 15.—A white woman, aged 54, with bronchiectasis, had a squamous-cell carcinoma of the lung. Prior to surgery, the electrocardiogram showed a left bundle branch block. On the fourth postoperative day following lobectomy, the heart developed an irregular rhythm which clinically was atrial fibrillation. Without treatment, it converted and the following day she had a normal electrocardiogram with disappearance of the branch block. No more arrhythmias were noted.

CASE 16.—A white man, aged 70, with hemangiopericytoma of the mediastinum was hypotensive following lobectomy. On the first postoperative day, he developed a rapid arrhythmia composed of multiple atrial ectopic beats. The following day, the heart was regular again. He did poorly and developed an effusion. Following atelectasis or pulmonary embolus, on the thirteenth postoperative day, the heart again was irregular for a while. In spite of digitalization, he continued downhill and died. Necropsy showed the heart to weigh 380 Gm., with normal myocardium. There were adhesions binding the left atria to the adjacent anterior superior left ventricle together with epicardial inflammation.

Ten of the 34 patients, or 29.4 per cent, surviving pneumonectomy, developed postoperative atrial arrhythmias. Six of the 42 patients, or 14.3 per cent, surviving lobectomies, developed a similar arrhythmia. Of the 77 surviving pulmonary surgery, 20.8 per cent developed arrhythmias.

DISCUSSION

Most of the cases occurred following right pneumonectomy; however, this probably represents the selectivity of a small series and is balanced by an increased incidence after left pneumonectomy in Humphreys' series.[4]

Most of the arrhythmias occurred in the first postoperative week and all occurred within the first month.[1-5]

Except for 2 patients, at the extremes of 37 and 70 years of age, all of the patients were in their sixth and seventh decades.

Type of Arrhythmia.—Sinus tachycardia was not considered to be an arrhythmia and was not included in the study.

All of the arrhythmias were atrial in nature. Four were multiple auricular ectopic beats occurring rapidly, with bursts and runs. One was a supraventricular tachycardia. The remaining eleven were atrial fibrillations. Ventricular ectopics were seen in only one of the patients.

Morbidity and Mortality.—The majority of the arrhythmias were fleeting in nature and tended to revert to normal rhythm spontaneously or aided by quinidine. In no case did a chronic arrhythmia develop. Symptoms often were marked with dyspnea, weakness, palpitation, and occasionally hypotension. Many of the arrhythmias coexisted with other complications; i.e., empyema, atelectasis, pneumonia, and pulmonary embolus, therefore allocating the exact etiology of the symptoms may not be accurate.

There was one death in this series which was felt to be due to the arrhythmia (Case 6). In this case, there was coronary artery disease without occlusion which was unsuspected prior to surgery.

Treatment.—All but 2 of the patients were treated with digitalis, which in the main was Cedilanid. In 5, there was slowing of the heart rate. Seven hearts spontaneously converted to normal rhythm shortly after the start of digitalization. Two continued to have runs of arrhythmia while the patients were on digitalis. One patient was not controlled by digitalization and continued on to failure and death.

Four patients required quinidine for conversion or stabilization of regular rhythm.

Auricular ectopic beats are less responsive to treatment than fibrillation.

Etiology.—In order to ascertain if possible the factors which are of etiological importance in these arrhythmias, the conditions which usually predispose toward arrhythmias were considered. Nearly all the patients had cardiac evaluation prior to surgery and, of those who had arrhythmias, only 2 had preexistent evidence of heart disease. One of these had no clinical evidence of heart disease but had left bundle branch block which disappeared after the arrhythmia. The other had pre- and post-thyrotoxic arrhythmias. One case showed right bundle branch block after the arrhythmia without evidence of heart disease.

Four of the patients died in the hospital and were examined at necropsy. One had a normal heart. One showed extensive coronary artery disease without occlusion but with subendocardial hemorrhage and cardiac dilatation. In one, there was pericardial effusion with right-sided dilatation. The fourth had atrioventricular adhesions together with local inflammatory reaction.

From the anatomic and clinical findings, we may conclude that the following cardiac factors *may* be of significance in the production of such arrhythmias: (1) Pre-existing paroxysmal fibrillation, (2) conduction defects due to unknown cause, (3) coronary artery disease, (4) pericardial effusion and right heart failure, and (5) localized atrial pericarditis.

Atrial fibrillation has no single known cause and no specific pathologic anatomy. There are no new factors exhibited in this study. Cardiac conditions in which this is prone to occur are well known and for the most part were absent in our cases and the others reported.[1-5]

Factors Which May Be of Significance.—

1. *The reduction in the pulmonary vascular bed which could cause elevated right-sided pressure and cor pulmonale:* Cournand has found that this elevation is usually rapidly adjusted and found only 2 cases with late elevation after pneumonectomy.[11] Atrial fibrillation and other atrial arrhythmias are not usually thought to be typical sequelae of cor pulmonale. However, in my experience, this is not unusual in other causes of cor pulmonale. Against this being the chief cause is the high incidence of arrhythmias in thoracic surgery, described by Currens and co-workers, where no pulmonary tissue is removed.[2] Catheterization studies were not done on any of our series and therefore the presence or absence of increased pressure can be only speculative.

2. *Infection and the inflammatory reaction of the neighboring structures:* In many of the cases, empyema or pneumonia became evident almost simultaneously with the arrhythmia and certainly there was inflammatory reaction in the surrounding tissue in most cases. The greater frequency with the more extensive procedure would favor this point as would the occurrence following esophageal surgery.[2]

3. *Hypoxia:* The nature of the operation tends to lower oxygen saturation levels and often the arrhythmia occurred after a complication which might further lower the ventilatory capacity. Iglauer and associates[8] feel that general hypoxia, experimentally, has nothing to do with the production of fibrillation. James and Geoghegan, on the other hand, have shown that localized cardiac anoxia will produce fleeting auricular arrhythmias.[10]

4. *Local inflammation of the atrium:* Atrial inflammation can occur, although it certainly cannot be the usual thing in these hearts. One of our cases appeared to have atrial pericarditis. The higher incidence of fibrillation following mitral surgery appears to reflect the inflammatory results of direct trauma on the distended atrium.[6]

5. *Vagal effect:* Bailey and Betts[1] had suggested that inflammation about the bronchial stump might cause vagal irritation and, by reflex, precipitate the fibrillation. It has been shown experimentally in man and animals that electrical, mechanical, and chemical stimulation of the vagus will produce atrial fibrillation in the sensitized heart.[7-9]

Incorporating the factors which may have some relation to the process, one could postulate the following theory: The atria, made irritable by hypoxia, arteriosclerosis, age, local reactive pericarditis, direct trauma, dilatation, hyperthyroidism, or other unknown factors may have one or many subthreshold ectopic foci which are kept inactive by the strong regular sinus rhythm. With either increase of the irritability or suppression of the sinus beat by vagal stimulation, the foci become threshold and arrhythmias, in the form of ectopic atrial systoles, atrial tachycardia, flutter or fibrillation, occur.

Prophylaxis: The high incidence of these arrhythmias, plus potential morbidity and mortality, leads to the conclusion that prophylaxis, if possible, should be exercised in every patient having thoracic surgery.

Black and Harken[6] have indicated that quinidine postoperatively had reduced their 75 per cent incidence of fibrillation after commissurotomy.

If the vagal effect in the mechanism of this arrhythmia exists, then the addition of anticholinergic drugs might potentiate the vagal effect of quinidine in preventing this complication.

It is, therefore, the purpose of this paper to suggest the routine use of quinidine and an anticholinergic drug, postoperatively, in all cases of thoracic surgery.

Since the majority of these patients can take oral medication immediately after surgery, the proposed schedule would be: (1) Quinidine 0.2 Gm. four times daily, with an added 0.2 Gm. of enteric-coated quinidine at bedtime, and (2) atropine 1 mg., four times daily (or other anticholinergic equivalent).

Such medication should be started on the first postoperative day and continued through the thirtieth postoperative day.

A constant expectant observation of the patient should be continued through the postoperative course and should arrhythmia occur, in spite of prophylaxis, rapid digitalization with Cedilanid or acetyl strophanthin should be instituted. If slowing does not occur, therapeutic doses of quinidine should be given without delay.

This report is intended to precede a future report of the success or failure of this prophylactic regime and perhaps will stimulate other studies of this problem.

SUMMARY

Sixteen cases of cardiac arrhythmias occurring in the postoperative course of pulmonary lobectomy and pneumonectomy are presented. This represents an incidence of 29.4 per cent, following pneumonectomy, and 14.3 per cent, following lobectomy, or a combined incidence of 20.8 per cent following pulmonary surgery in 77 cases. Possible etiological factors are discussed and because of the incidence, morbidity, and possible mortality, routine prophylaxis with quinidine and atropine is suggested.

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INSERTION OF A HUFNAGEL VALVE FOR AORTIC INSUFFICIENCY COMPLICATED BY DEVELOPMENT OF A FALSE ANEURYSM

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In 1949, Hufnagel first reported on the experimental use of a plastic valve replacement for aortic insufficiency in dogs.[1] In 1953, he reported on the use of such a prosthesis in human beings.[2] A supplemental report in 1954, and again in 1955,[3, 4] gave further evidence that this approach to correction of the management of aortic insufficiency is practical and beneficial.

Fixation of the valve in the aorta is accomplished by multitoothed circumferential nylon rings. The intermittent circumferential pressure exerted by this type of ring was found to be necessary by Hufnagel to prevent necrosis and subsequent breakdown of the union between the aorta and the valve.

Complications have arisen. Hufnagel[4] mentions thromboembolic phenomena in their early experience but not in recent cases. One case of aneurysm formation was reported by him, apparently believed to be due to improper fit of the valve, the valve being smaller than the aorta. The subsequent course of this patient is not mentioned. Lord and Stone[5] report, in 1955, a patient of theirs who developed a false aneurysm with ultimate death. The following case report in many respects is quite similar to that reported by Lord and Stone.

CASE REPORT

T. O., a 48-year-old Negro man, was admitted on Feb. 1, 1955, for the sixth time for treatment of cardiac failure. The periods of decompensation had become more frequent in recent years and he had developed angina pectoris so severe that he was unable to walk more than five to ten steps. His rheumatic fever dated back fifteen or twenty years. Examination revealed a well-developed, well-nourished Negro man, very slightly dyspneic at rest. Blood pressure was 160/50 in the arms and 170/50 in the legs. Neck veins were distended. The lungs revealed a few scattered moist râles. Cardiac rhythm was regular. A Grade 3 systolic murmur was heard in the primary aortic area and transmitted into the neck. A Grade 3 diastolic murmur was audible along the left sternal border. A harsh, rumbling Grade 4 diastolic murmur and a systolic murmur of less intensity were present at the apex. All peripheral signs of aortic insufficiency were present.

Chest x-ray (Fig. 1) and fluoroscopy revealed a cardiothoracic ratio of 0.65. In the left anterior oblique position, the right ventricle was slightly enlarged and the left ventricle was greatly enlarged, barely clearing the spine in the lateral position. There was very slight posterior displacement of the barium-filled esophagus, indicating insignificant left

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atrial enlargement. Electrocardiograms were interpreted as showing left ventrical hypertrophy. Serologic tests were negative. Routine laboratory work was within normal limits. It was our impression that the mitral disease was of minimal degree and that the aortic regurgitation was of primary importance.

On Feb. 23, 1955, a Hufnagel valve was inserted. This was carried out through a left thoracotomy incision in the fifth intercostal space. The aorta, just distal to the origin of the left subclavian artery, was mobilized by ligating six pairs of intercostal arteries and one bronchial artery. After cross-clamping above and below the proposed point of insertion of the valve, an attempt was made to insert a No. 3 valve but the friable aorta began to tear and a No. 2 valve was rapidly inserted. This proved to be slightly smaller than the aorta. When the clamps were removed there was some leakage both at the upper and lower ends of the valve and the aorta was again cross-clamped. With the aorta again clamped,

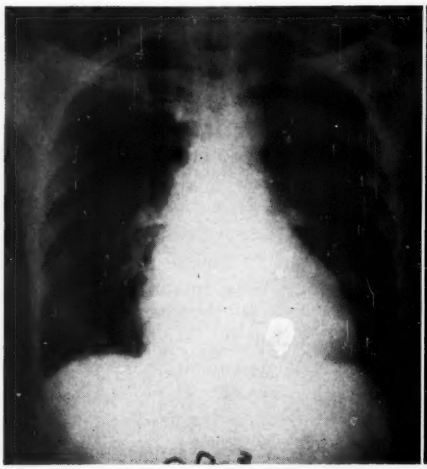

Fig. 1.

Fig. 2.

Fig. 1.—Posteroanterior view of chest, preoperative, showing left and right ventricular enlargement.

Fig. 2.—Posteroanterior view of chest, thirteen days postoperatively, showing evidence of operative intervention and perihilar density interpreted as pleural reaction.

No. 3-0 silk sutures were tied loosely on the valve edge of the aorta just distal to the proximal constriction ring and just proximal to the distal constriction ring. This controlled the leakage satisfactorily. Gelfoam was wrapped around the valve and placed between the valve and the vertebral bodies. Seventeen minutes were required during the first period of cross-clamping and an additional four minutes during the second period.

He tolerated the operation well. Postoperatively, he was put on anticoagulant therapy for ten days. Digitoxin was continued in maintenance dose and penicillin was given for one week. He did extremely well. Chest drainage was 300 c.c. in twenty-four hours, and the tube was removed on the second postoperative day. A low-grade fever subsided on the tenth postoperative day. The blood pressure stabilized rapidly at approximately 130/0 mm. Hg in the arms and 160/90 mm. Hg in the legs.

Chest x-ray (Fig. 2), taken on the thirteenth postoperative day, revealed a haze in the left mid-lung field and was interpreted as a pleural reaction. This remained unchanged during the time of his hospitalization. Approximately three weeks after surgery, he began

to run a sustained temperature with spikes to 101° F. associated with left chest pain. Exploratory thoracentesis, because of his fever, was carried out on three occasions but no empyema was found. The fever subsided in approximately one month and he was discharged.

The angina was improved to the extent that he could walk a block without having recurrence of pain and he was less dyspneic. He did well until approximately six months after surgery when he began to complain of a cough, slightly blood-streaked sputum, left posterolateral chest pain, fever, weakness, and anorexia.

He was readmitted on Sept. 28, 1955, with a temperature of 99.4° F. and a complaint of left posterolateral chest pain. X-rays of the chest revealed slight increase in haze in the left mid-lung field. The cough was productive of blood-tinged sputum. Blood pressure was 135/0 mm. Hg in his arms and 165/90 in the legs. The Hufnagel and heart valve sounds were unchanged. He improved for the first two or three days but on the eighth day after admission he complained of increasing chest pain and coughed up a considerable quantity of bright red blood. The next day, when he was seen at the request of the Medical Service, he was raising blood, had moderately severe dyspnea and complained bitterly of left chest pain. A chest x-ray (Fig. 3) showed considerable increase in the

Fig. 3.—Posteroanterior view of chest, seven months postoperatively, showing increase in mid-lung field density, interpreted as a false aneurysm.

size of the mid-lung field density. A false aneurysm was suspected and thoracentesis in the valve area yielded bright red blood. Approximately ten minutes after the aspiration, the patient began to cough up more blood and rapidly asphyxiated due to blood in the tracheobronchial tree.

Post-mortem examination revealed a false aneurysm apparently arising from the posterior aspect of the proximal end of the valve. The aneurysm, which measured approximately 10 cm. in length, consisted of laminated clots. The lateral wall of the aneurysm was composed of the superior segment of the left lower lobe, while the aorta and valve formed the other walls. The false aneurysm communicated with the aorta by means of a one millimeter channel passing beneath the proximal constriction ring on its posterior aspect (Figs. 4*A* and 4*B*). No large bronchial communication with the aneurysm existed but there were several small bronchial openings into this aneurysm.

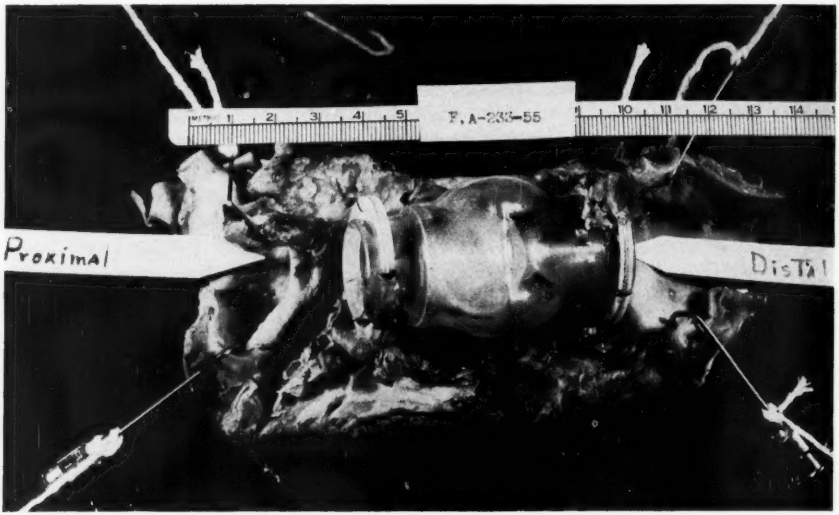

Fig. 4A.—Post-mortem specimen showing Hufnagel valve in the segment of aorta and a surrounding hematoma.

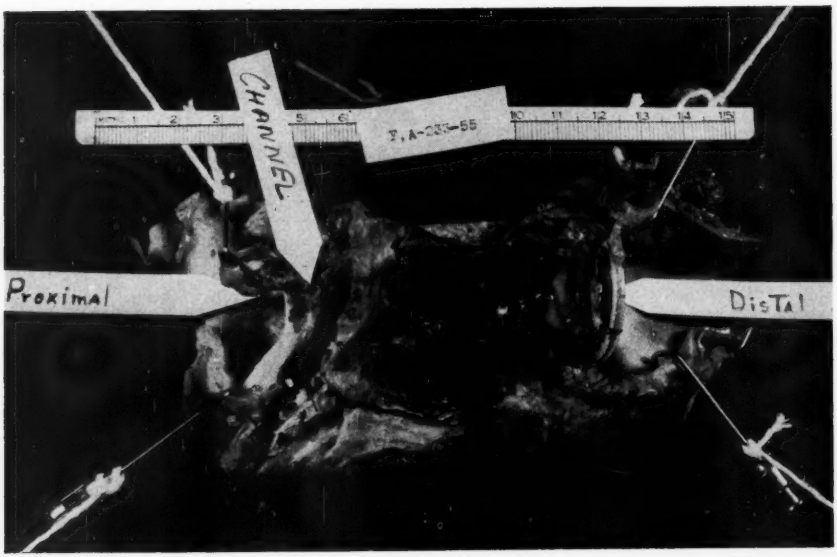

Fig. 4B.—Post-mortem specimen with Hufnagel valve removed. The one millimeter channel marker is slightly to left of the channel in this photograph.

DISCUSSION

Insertion of a Hufnagel valve in a 48-year-old Negro man, suffering from aortic insufficiency, has been reported. Insertion of the valve was complicated by difficulty of accurate fit, a No. 3 valve being too large while a No. 2 valve was found to be slightly too small. It was necessary to control the slight leak by use of lightly placed silk sutures. The postoperative course was complicated by some fever. He was considerably improved for seven months and then died of asphyxia due to leakage of a false aneurysm into the superior segment of the left lower lobe. The difficulty of accurate fit is great and may be exaggerated in the older arteriosclerotic aorta which, because of its decreased elasticity, requires a much more accurately fitting valve. Range of selection might be improved by the addition of a size between the No. 2 and No. 3 valves.

It was our impression from the post-mortem findings of a small one millimeter channel lying beneath the proximal constriction ring that the cause of the aneurysm was due to slight wrinkling beneath the ring as a result of the improper valve fit. Hufnagel mentions one case of aneurysm formation due to the placement of a valve smaller than the aorta but suggests that this was due to the aorta bulging over the ring and coming in contact with it, leading to erosion of the aorta and aneurysm formation. The case reported by Lord and Stone[5] suggests that their difficulty was due to insertion of a valve smaller than the aorta; they, however, apparently felt that necrosis at the site of the proximal constriction ring was the main contributing cause. By gross and microscopic examination of sections taken beneath the proximal constriction ring, we do not feel that necrosis of the aorta was a cause in our case. It is felt that the aneurysm was present from the immediate postoperative period but that the patient did not experience his final difficulty until break through into the tracheobronchial tree occurred, leading to his death from asphyxiation.

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MASSIVE ATELECTASIS OF THE LEFT LUNG IN CHILDREN WITH CONGENITAL HEART DISEASE

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MOST deaths from the more severe intracardiac forms of congenital heart disease occur in the first year of life. Recently, techniques have been developed enabling the correction of these grave defects. As a result of these advances, a number of infants, critically ill with congenital heart disease, are now being admitted for curative surgery in the University of Minnesota Hospitals.

Apart from the inherent technical problems associated with operations upon these small children, the postoperative period has at times presented unusual difficulties. Respiratory complications have been a major concern when characterized by viscid secretions and bronchiolitis. These respiratory difficulties have been accentuated by the necessity for a bilateral thoracotomy to provide an adequate surgical exposure.

A too frequent complication which we have encountered has been the appearance of atelectasis of the left lung, occurring either before or after surgery. Even more often, we have noted the existence of transient episodes of atelectasis in the left lower lobe and at times this has progressed to a massive collapse of that lung. In other cases where some difficulty was noted in the ease of expansion of the left lung during the operation, massive atelectasis has occurred in the postoperative period.

Eight representative cases of this syndrome, which have been recognized during the past fifteen months, are described below. Five of these patients had ventricular septal defects, one an aorticopulmonary window, another a patent ductus arteriosus with coarctation of the aorta and one had total anomalous pulmonary venous return to the right atrium. In 5 cases, atelectasis was present prior to surgical correction of the defect. In 3, massive collapse of the left lung appeared for the first time during the postoperative period.

CASE REPORTS

Preoperative Atelectasis

CASE 1.—D. M. (Fig. 1), a 5-week-old female, exhibited dyspnea from birth. Cardiomegaly and hepatomegaly were present and associated with a systolic murmur and thrill

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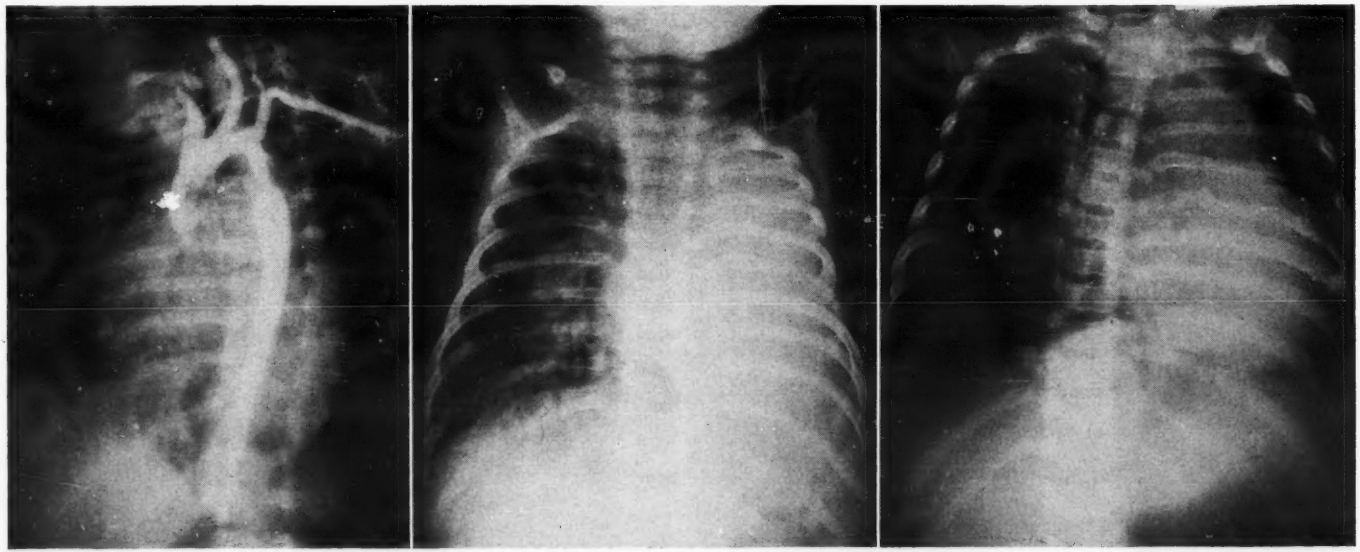


A. *B.* *C.*

Fig. 1.—Case 1. *A*, Retrograde aortogram demonstrating coarctation of aorta. Ductus arteriosus not visualized. Lung well expanded at this time. (Jan. 22, 1956.)

B, (Jan. 24, 1956.) Massive atelectasis left lung. Mediastinum herniated to left.

C, (May 3, 1956.) Three months postoperative. Partial re-expansion of left lung.

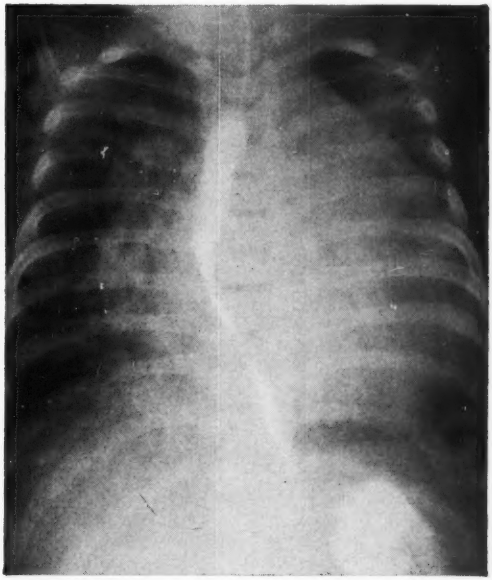
over the precordium. The femoral pulses were not palpable. Retrograde aortography revealed coaretation of the aorta and a patent ductus arteriosus proximal to the narrowed segment. The patient's condition deteriorated following the appearance of total atelectasis of the left lung. On Jan. 25, 1956, emergency thoracotomy was performed. The left lung was completely collapsed and could not be expanded. A large patent ductus arteriosus was transected. The coarcted segment was resected and the aorta anastomosed. Postoperatively, the patient improved slowly and was discharged from the hospital in three months. Atelectasis of the left lung still persisted at this time. Re-examination a week after discharge revealed partial re-expansion of the left lung.

CASE 2.—M. G. (Fig. 2), a 4-month-old female, was admitted on Jan. 30, 1956, for repair of a ventricular septal defect. There had been repeated bouts of congestive heart failure with hepatomegaly and peripheral edema. Cardiac catheterization had revealed a large left-to-right shunt at the ventricular level. The pulmonary artery pressure was 76/40 mm. Hg and femoral artery saturation was 86 per cent. She experienced transient episodes of atelectasis of the left lower lobe and five days following this admission massive atelectasis of the lung occurred on the left. The next day the ventricular septal defect was repaired under direct vision through a bilateral anterior thoracotomy incision, using the artificial oxygenator.[2] The left lung was collapsed and could not be re-expanded despite the effects of the anesthesiologist. Postoperatively, the patient had severe respiratory difficulty. The following day, pulmonary edema in the remaining lung eventuated in death. Post-mortem examination revealed anatomic repair of the septal defect. The left lung was atelectatic with evidence of compression of the left main bronchus by the overlying pulmonary artery.

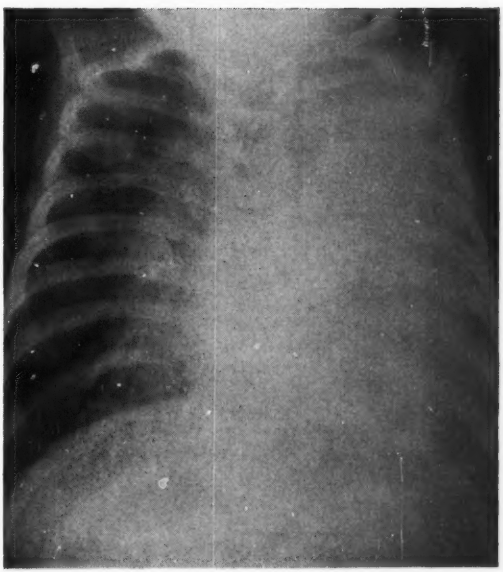
CASE 3.—D. H., a 16-month-old male, weighing 6 Kg., had had repeated upper respiratory infections since his birth. Cardiac catheterization and angiocardiography established the diagnosis of total anomalous pulmonary venous return to the right atrium. Pulmonary artery pressure was 50/19 mm. Hg. The patient's condition was poor. On June 1, 1956, massive atelectasis of the left lung appeared. Surgery was performed on June 4, 1956, through a bilateral anterior thoracotomy incision. The left superior pulmonary vein drained in the left innominate vein while the remainder of the pulmonary veins emptied into the right atrium via the coronary sinus. Utilizing the artificial oxygenator, a small foramen ovale defect was enlarged in the direction of the coronary sinus. A sheet of compressed polythene sponge was sutured over the coronary sinus and interatrial defect, thus directing the blood from the coronary sinus into the left atrium. Postoperatively, the patient did moderately well. Atelectasis of the left lung persisted. Forty-eight hours following surgery, acute pulmonary edema of the right lung resulted in death. Post-mortem examination revealed findings as noted at surgery. The left pulmonary artery compressed the left main bronchus with resultant bronchial occlusion.

CASE 4.—(Fig. 3) N. T., an 8-month-old boy, was admitted on Feb. 8, 1956, following the detection of a precordial murmur and thrill. Cardiac catheterization revealed a ventricular septal defect with right ventricular pressure of 66/2 mm. Hg. Chest x-ray revealed moderate right ventricular and left atrial enlargement. Furthermore, there was a small amount of atelectasis of the left lower lobe. Surgery was deferred temporarily since he was developing normally. On June 12, 1956, he was readmitted in acute distress. There was absence of breath sounds over the left chest except at the left apex. Chest x-ray revealed complete atelectasis of the left lower lobe with emphysema of the left upper lobe. Three days later complete atelectasis ensued on the left. Atelectasis of the left lung persists. Surgical repair of the ventricular septal defect is planned for the immediate future.

CASE 5.—H. M., a one-month-old female, was admitted on April 6, 1956, with evidence of congestive heart failure. Cardiac catheterization revealed a ventricular septal defect and a pulmonary artery pressure of 64/42 mm. Hg. After digitalization and diuretics, cardiac

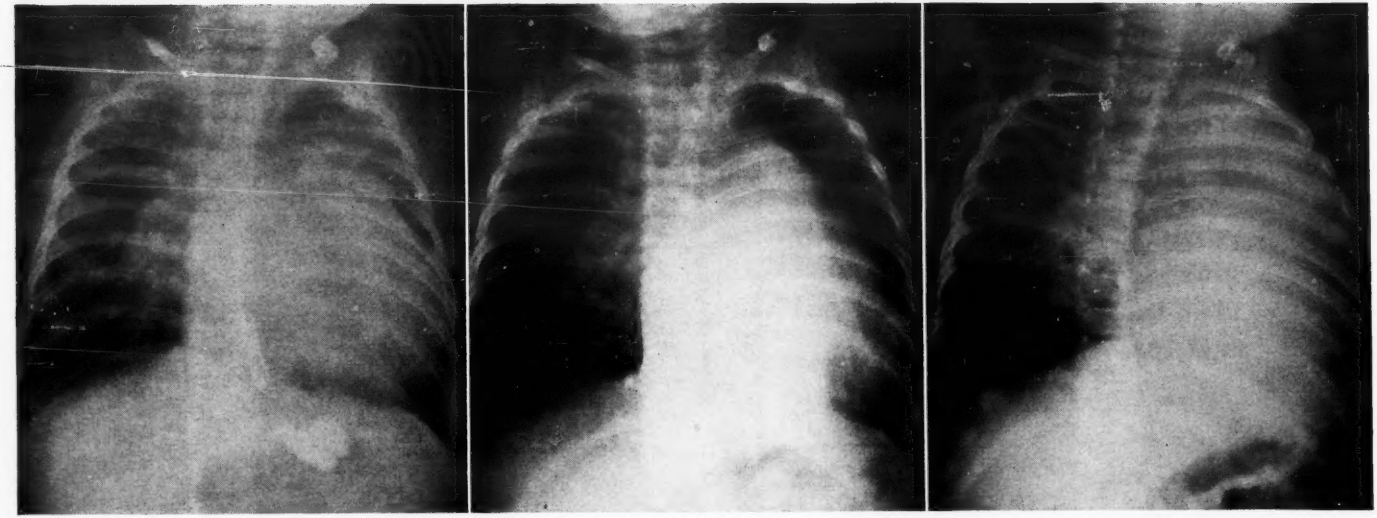


A.



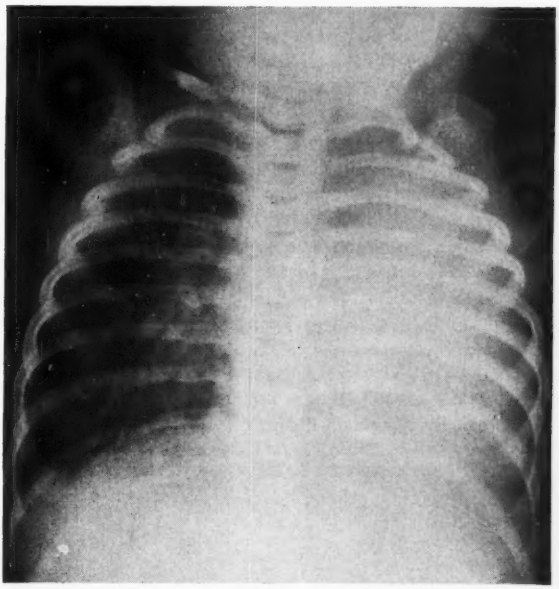
B.

Fig. 2.—Case 2. *A*, (Jan. 3, 1956). Slight atelectasis involving left lower lobe. *B*, (Feb. 4, 1956). Massive atelectasis, left lung.

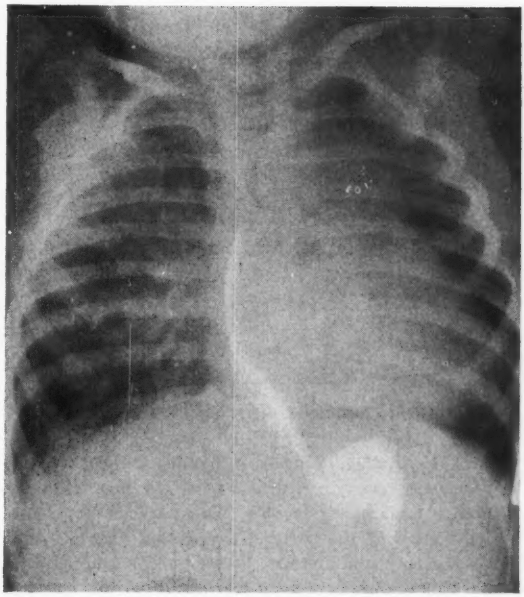


A. *B.* *C.*

Fig. 3.—Case 4. *A*, (Feb. 8, 1956.) Atelectasis involving left lower lobe. *B*, (June 12, 1956.) Atelectasis left lower lobe and emphysema of left upper lobe. *C*, (June 15, 1956.) Massive atelectasis of left lung.



A.



B.

Fig. 4.—Case 6. *A*, (April 28, 1955.) Massive atelectasis of left lung appearing one month postoperatively.
B, (Nov. 1, 1955.) Re-expansion of left lung.

compensation was re-established. However, concomitantly, aeration of the left lung decreased. Initially, left lower lobe atelectasis appeared. Subsequently, atelectasis extended to involve most of the left upper lobe. This total atelectasis has persisted. At present the patient is awaiting surgical correction of the ventricular septal defect.

Postoperative Atelectasis

CASE 6.—B. H. (Fig. 4), a 7-month-old female, was admitted for repair of a ventricular septal defect. Cardiac catheterization revealed a left-to-right shunt at the ventricular level with a pulmonary artery pressure of 80/40 mm. Hg. On March 25, 1955, utilizing cross circulation,[1] the ventricular septal defect was repaired under direct vision through a right ventricular cardiotomy. In addition, a 2 cm. atrial septal defect was repaired through a simultaneous right atrial cardiotomy. The patient did well postoperatively. However, complete atelectasis of the left lung supervened. Bronchoscopy revealed obstruction of the left main-stem bronchus just above the origin of the left upper lobe, due to edema and extrinsic compression. Complete atelectasis persisted for two months. Subsequently a slow re-expansion of the lung has transpired. On Dec. 1, 1955, cardiac catheterization revealed absence of any left-to-right shunt and a pulmonary artery pressure of 18/0 mm. Hg.

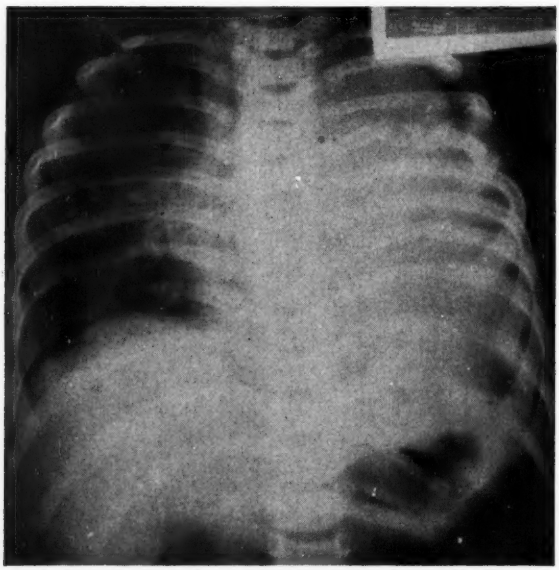


Fig. 5.—Case 8. Massive atelectasis of left lung, twenty-four hours postoperatively.

CASE 7.—M. F., an 11-month-old female, was admitted for repair of a ventricular septal defect. The pulmonary artery pressure was 70/32 mm. Hg. On April 3, 1956, through a bilateral anterior thoracotomy incision, the ventricular septal defect was repaired under direct vision with the aid of the artificial oxygenator. During surgery, the left lung aerated poorly and expanded only with difficulty. Twenty-four hours later, massive atelectasis of the left lung ensued. This persisted for four days despite vigorous tracheobronchial aspiration. Subsequently, partial re-expansion of the left upper lobe had taken place. The patient did moderately well during the next week but eventually succumbed to a septicemia,

secondary to a suppurative thrombophlebitis involving the inferior vena cava. The portal of entry for these organisms was the saphenous vein cutdown which had been left in for a longer than usual period because of these respiratory complications.

CASE 8.—A. F. (Fig. 5), an 8-month-old female, had a precordial systolic murmur first noted shortly after birth. Development was slow, marked by repeated upper respiratory infections, and an episode of congestive heart failure. The findings at cardiac catheterization were compatible with the diagnosis of patent ductus arteriosus. On March 26, 1956, exploratory thoracotomy revealed the presence of a large aorticopulmonary window. The ductus arteriosus had been obliterated. On April 17, 1956, through a bilateral anterior thoracotomy incision, the aorticopulmonary window was divided and the aorta and pulmonary artery repaired. The artificial oxygenator was used in conjunction with retrograde perfusion of the coronary sinus. During surgery, it was noted that the left lung expanded only with difficulty. The patient did well for twenty-four hours at which time massive atelectasis of the left lung occurred. Progressive respiratory difficulty ensued. Accordingly, the patient was curarized, an endotracheal tube inserted and attached to a positive and negative phase respirator. Despite these procedures, aeration of the left lung was minimal. Although the patient gradually improved enough so that in three days she was handled with spontaneous respiration, unfortunately, she developed a progressive, diffuse, bronchiolitis and, despite reinstitution of the respirator, she died on the tenth day. Autopsy revealed atelectasis of the left lung with compression of the left main bronchus by the large pulmonary artery.

DISCUSSION

This precise form of massive unilateral atelectasis we have seen only in infants with symptomatic congenital heart disease. Furthermore, this condition has selectively involved the left lung. This pulmonary problem has not occurred with the reduced pulmonary blood flow that exists in the tetralogy of Fallot defect. All of these atelectatic patients have had an increased blood flow through the lungs and serious degrees of pulmonary hypertension. At surgery, the pulmonary artery was observed to be large, tense, and pulsatile.

The mechanism for the production of atelectasis of the left lung we have ascribed to compression of the left main bronchus by an enlarged left pulmonary artery. This observation has been made not only during operations but at post-mortem examinations also. Furthermore, bronchoscopy has revealed obstruction which is extrinsic and involving the left main-stem bronchus at the site where the left pulmonary artery crosses it. A similar type of compression by an enlarged left atrium has not been noted. In further emphasis of this point, Case 3 had an abnormally small left atrium in association with total anomalous pulmonary venous return to the right atrium, yet still developed left-sided atelectasis. Massive atelectasis involving the right lung has not been observed. This may well be due to the altered relationship between the right pulmonary artery and its bronchus. Moreover, here only the right intermediate bronchus is crossed by a pulmonary artery which has already divided. Certainly, the fact that all of these hearts were massively enlarged and that most of this encroachment was at expense of the left lung contributed to this localization.

The prevalence of this condition in infancy is probably related to the small lumen and soft nature of the bronchus. A similar degree of severe pulmonary

artery enlargement in older patients does not result in a corresponding state of bronchial obstruction.

The atelectasis when studied for pathologic relationship has been found to be acquired in all cases. In no instance has there been evidence of pulmonary hypoplasia. In each case, the lung has been fully expanded at some time prior to the appearance of atelectasis.

A review of chest films in patients with congenital heart disease producing pulmonary hypertension reveals varying degrees of left bronchial obstruction. A high proportion of infants with pulmonary hypertension show minor to major degrees of atelectasis and/or obstructive emphysema. More commonly this appears to begin with involvement of the left lower lobe which progresses in some cases to massive atelectasis of that entire lung. Suppurative bronchopneumonia of this lung, associated with partial bronchial obstruction, has complicated the final picture at times.

Respiratory infections uncomplicated by bronchial obstruction are frequently observed in the presence of increased pulmonary blood flow. It is important to differentiate those from these cases of atelectasis with or without superimposed infection. Krabbenhoft and Evans[3] described a variegated group of patients who at post-mortem examination had pulmonary atelectasis or emphysema and a cardiac anomaly. Case 3 of this group is similar to those described by them. They attribute the pulmonary changes to chondromalacia of the bronchi or to muco-viscoidosis. In our cases the specific localization of the bronchial obstruction appears as a more likely cause than these factors.

The appearance of partial or total collapse of the left lung has been considered an indication for, rather than a contraindication to, definitive correction of the cardiac anomaly since spontaneous resolution of the atelectasis has only been observed by us to follow a curative operation. Admittedly, the surgical mortality will be increased in the presence of pulmonary atelectasis; nevertheless, surgical repair seems to offer the only hope of salvaging such a patient's life. The onset of radiologically visible obstructive emphysema in the left lung represents an urgent indication for curative surgery before massive collapse occurs. Although several of these infants with massive atelectasis of the left lung and left-to-right shunts had systemic aterial oxygen saturations in the 80 per cent range, this degree of desaturation under these conditions we believe does not preclude a curative operation. A considerable volume of partially desaturated blood probably moves through the nonaerated lung.

Moreover, in none of these 5 patients, with atelectasis prior to surgery, was it possible at thoracotomy to effect even the slightest visible expansion in this atelectacic lung even after correction of the shunt and with the maximal positive pressures physiologically safe. This is in part explicable by an observation which we have confirmed many times. With serious degrees of pulmonary hypertension, the vascular changes, primarily responsible, regress slowly (over a matter of weeks and months). Therefore, the immediate change in the pulmonary pressures following a completely corrective anatomic procedure may

be negligible. In fact, we have actually measured during the operation increases in the pulmonary artery pressure after corrective surgery. Although patients with this type of case ultimately may recover, such an increase in pulmonary pressure is an ominous prognostic sign.

Certainly, the surgeon and anesthesiologist must protect to the utmost the remaining functional lung (right) and avoid damaging it through persistent attempts at re-expanding the left lung through indiscreet application of unphysiologically high positive pressures capable of rupturing the intact respiratory system.

SUMMARY

1. Eight cases of massive atelectasis of the left lung in infants with congenital heart disease are described. All of these cases had increased pulmonary blood flow and pulmonary hypertension.

2. Evidence is presented relating this syndrome to compression of the left main bronchus by the left pulmonary artery.

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ARTERIAL PATTERNS OF LEFT UPPER LOBE

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INTRODUCTION

THE arterial supply of the left upper lobe is one of the most intricate and interesting and presents a wide variety of patterns which has occasioned considerable study and discussion in the literature. These complicated distributions can endanger various segments during surgery. It would not be irrelevant to conjecture that one or more segments could be easily and inadvertently deprived of circulation. True, the unintentional deprivation of a small segmental artery or branch here or there might not be of catastrophic significance or even detected. Ligation of even major pulmonary arteries to a lobe or lobes usually does not result in gangrene so long as the bronchial artery supply and the pulmonary venous return is intact.[4] Therefore, the absence of obvious postoperative pulmonary complications does not prove that the resection has been perfect. Such unintentional ligation can, however, cause a useless and unnecessary shrinkage of the lung concerned and deprive it of respiratory and space-filling functions postoperatively. For these reasons, it was thought that further detailed anatomic description of a modest number of anatomic specimens might be interesting and helpful.

Boyden and Hartmann[1] in fifty left upper lobes found from four to eight segmental arteries. An anterior origin of arterial rami to the anterior segment was consistently present. The apical segment artery had an anterior origin in 38 per cent and the lingula artery (or part) an anterior origin in 22 per cent. The apical artery had a superior origin in 40 per cent. The posterior segment artery arose from the posterosuperior aspect of the left pulmonary arch in 42 per cent. The interlobar aspect of the lobe showed predilection for segmental arteries to cross intersegmental planes. Posterior subsegment arteries of anterior and posterior segments varied considerably in origin. In 36 per cent, the interlobar origin of the lingular arteries and those for the posterior subsegment of the anterior segment was from a common stem.

Barrett, Day, and Tuttle[3] charted the arterial distribution immediately after surgery in 100 cases of left upper lobectomy or multiple segmental resection. In 87 per cent, there were three to five arteries to the lobe, with the greatest number of patients having four. The limits were two to seven. The anterior segment received more than one artery in less than 10 per cent. In

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The views expressed by the author are his own and do not necessarily reflect the opinion of the Veterans Administration.
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over 50 per cent, the apicoposterior supply was one of four dominant patterns. In the remaining 43 per cent, there were eighteen different patterns. The pattern to the apicoposterior segment was the most variable of all.

They found for the lingula segment one artery in 89 per cent, two arteries in 10 per cent and three arteries in one per cent. In two-thirds of their cases, the lingula artery or arteries arose at or below the level of origin of the superior segment artery to the lower lobe. This would necessitate, in lower lobe resection, the ligation of the superior segment artery separately and then the main arterial trunk below the lingular artery origin. The level of origin of the lingular artery in relation to the superior segment artery was as follows: above it in 33 per cent, at the same level in 33 per cent, and below it in 34 per cent.

In only about one-sixth of their cases did the lingular artery or arteries supply segments other than the lingula. In only 8 per cent did the anterior segment receive even part of its supply from lingular arteries. In 4 per cent, the lingular artery sent a branch to a basal segment. In only 10 per cent was the apicoposterior trunk the first branch of the pulmonary artery. Conversely, in over 80 per cent the first branch was found to be an anterior or apicoanterior artery. They did not find the lingular artery to be a significant source of supply to the anterior segment.

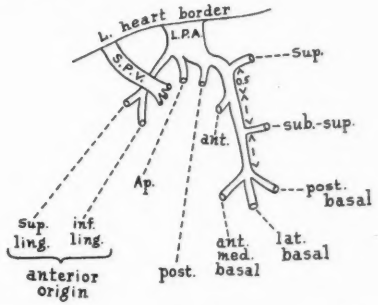


Fig. 1.—An anterior origin of lingular arteries. (Numbers are in centimeters.)

MATERIAL

This comprised anatomic dissections of 25 adult male post-mortem heart-lung specimens, some of which have previously been depicted.[4] Patterns not previously shown are found in Figs. 1 to 16.

RESULTS

The left upper lobe was supplied by three to six arterial stems arising from the left pulmonary artery. Eight per cent of specimens had three stems, 32 per cent had four stems, 36 per cent had five stems, and 24 per cent had six stems. The first branch of the left pulmonary artery gave branches to the various segments as follows: anterior in 52 per cent, anterior-apical in 12 per cent, apical in 8 per cent, lingula in 8 per cent, anterior-lingula in 8 per cent, posterior in 4 per cent, anterior-posterior in 4 per cent, and anterior-lingula-posterior in 4 per cent.

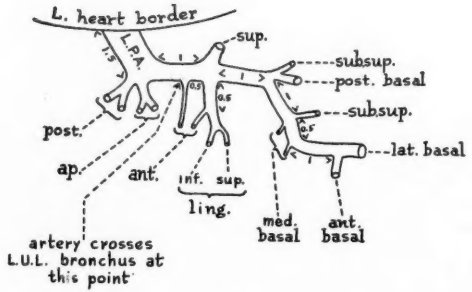
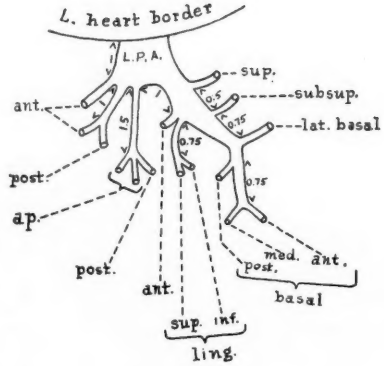


Fig. 2.



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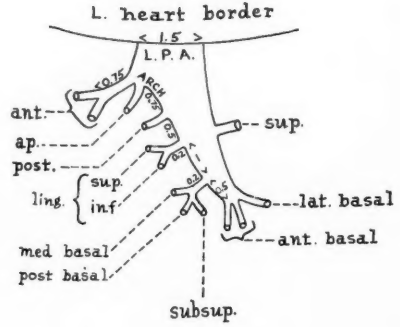


Fig. 4.

Fig. 2.—Lingular artery supplies anterior segment.
Fig. 3.—Anterior segment has an anterior and interlobar supply. An interlobar stem bifurcates to supply anterior and lingula segments.
Fig. 4.—Anterior origin of anterior and apical segments.

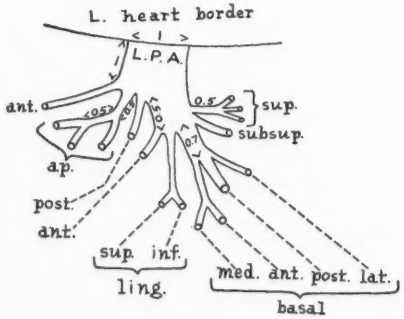
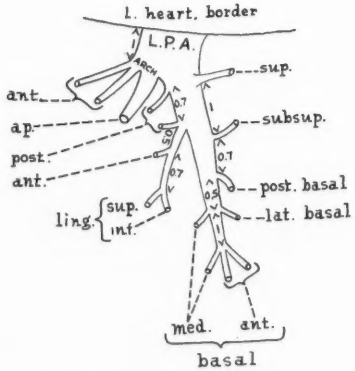


Fig. 5.



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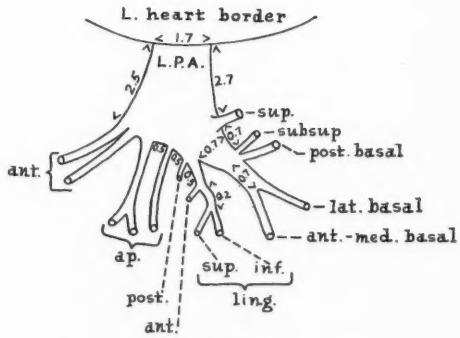
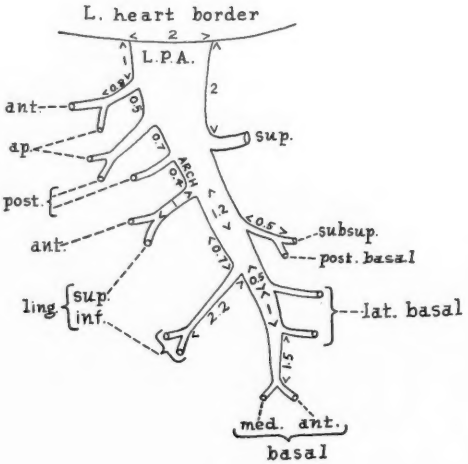


Fig. 7.

Fig. 5.—Lingular artery is one branch of a quintuplate pattern which supplies also anterior and lower lobe segments.

Fig. 6.—Lingular artery forms a bifurcate pattern with a posterior segment artery and gives a branch to anterior segment. Anterior segment also has an additional anterior supply.

Fig. 7.—Lingular artery supplies the anterior segment. Anterior segment also has an additional anterior supply.



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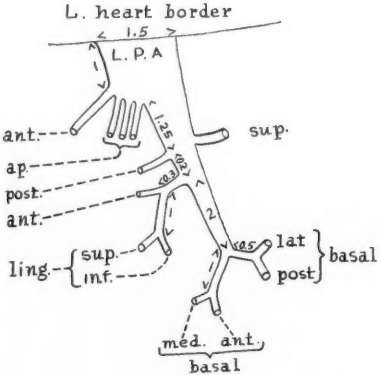


Fig. 9.

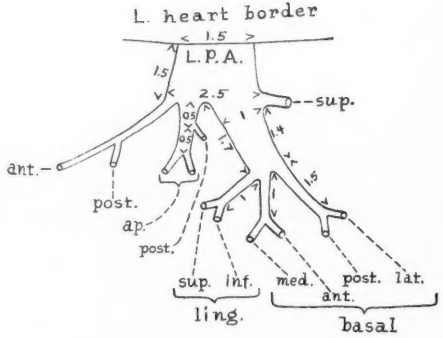
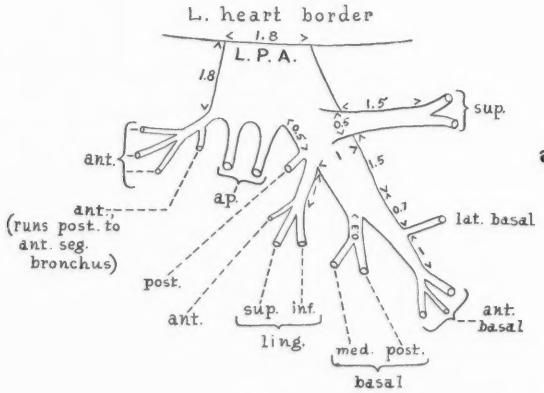


Fig. 10.

Fig. 8.—Superior lingular artery is one branch of a bifurcation which also supplies anterior segment. Anterior segment has an additional anterior supply.

Fig. 9.—Lingular artery is one branch of a bifurcation which also supplies the anterior segment. Anterior segment also has an anterior supply.

Fig. 10.—Lingular artery is one branch of a trifurcation which also supplies the basal segments of lower lobe. Anterior segment has an anterior supply only.



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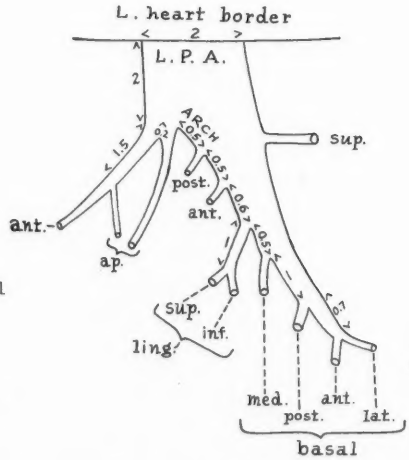

Fig. 12.

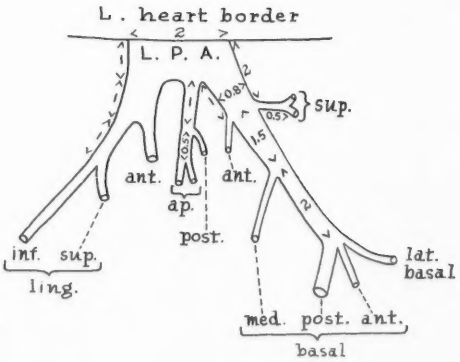

Fig. 13.

Fig. 11.—Lingular artery supplies anterior segment. Anterior segment also has an anterior supply.
Fig. 12.—Anterior segment has an anterior and interlobar supply.
Fig. 13.—Lingular artery is anterior and forms one branch of a bifurcation which also supplies anterior segment.

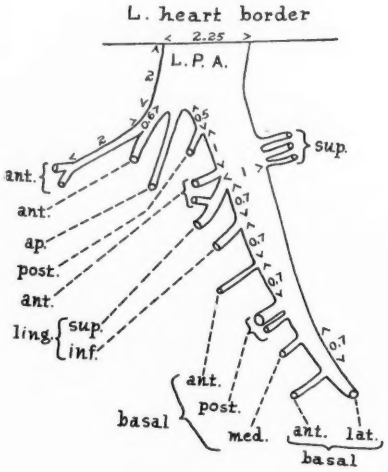

Fig. 14.

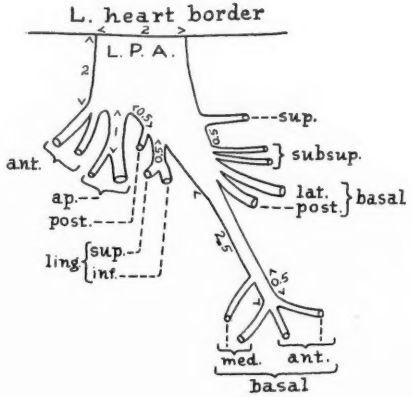

Fig. 15.

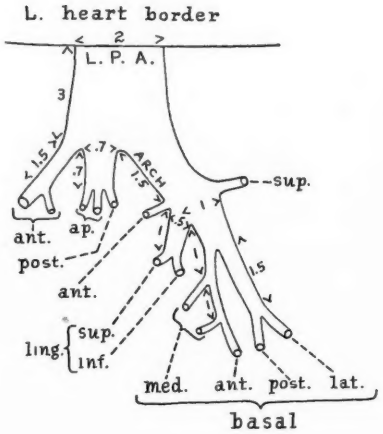

Fig. 16.

Fig. 14.—Lingular artery supplies anterior segment. Anterior segment has an anterior and interlobar supply.
Fig. 15.—Anterior segment is supplied entirely by an artery arising anteriorly.
Fig. 16.—Anterior segment has an anterior and interlobar supply.

In 24 per cent, an artery for the lingula segment arose anteriorly near the hilus. In 84 per cent, an artery for the anterior segment arose directly or indirectly from an anterior artery near the hilus. The anterior segment received more than one artery in 76 per cent. The apicoposterior segment was supplied solely by a common trunk or two separate trunks arising directly from the left pulmonary artery in 16 and 40 per cent, respectively. *Further:*

a. A lingular artery was one branch of a bifurcation which also supplied the anterior segment in 6 or 24 per cent
b. A lingular artery was one branch of a bifurcation which also supplied the posterior segment in 1 or 4 per cent
c. A lingular artery was one branch of a trifurcation which also supplied the anterior segment in 1 or 4 per cent
d. A lingular artery was one branch of a trifurcation which also supplied the lower lobe basal segments in 1 or 4 per cent
e. A lingular artery was one branch of a quintuplate pattern which also supplied the anterior and lower lobe segments in 1 or 4 per cent
f. A single arterial stem for the lingula segment arose from the left pulmonary artery in 19 or 76 per cent
g. A lingular artery gave a true branch to the anterior segment in 6 or 24 per cent
h. A lingular artery gave a true branch to the anterior and posterior segments in 1 or 4 per cent
i. A lingular artery had an exclusively anterior origin in 3 or 12 per cent
j. A lingular artery arose both anteriorly and in the interlobar fissure in 3 or 12 per cent
k. A lingular artery in the interlobar fissure arose at the level of (40 per cent) or below (48 per cent) the origin of the superior segment artery in 22 or 88 per cent

SUMMARY

In 25 dissected left upper lobes, a lingular artery gave a true branch to the anterior segment in six instances, or 24 per cent. A lingular artery gave branches to both anterior and posterior segments in an additional instance, making a total of 28 per cent. A lingular artery arose from a common stem with an anterior segment artery in 24 per cent. A lingular artery arose from a common stem with a posterior segment artery in 4 per cent. A single arterial stem for the lingular arising from the left pulmonary artery was found in 76 per cent. In 24 per cent, an artery for the lingula segment arose anteriorly near the hilus. In 12 per cent, a lingular artery had an exclusively anterior origin. In 12 per cent, a lingular artery arose both anteriorly and in the interlobar fissure. When a stem for the lingula was interlobar, it arose at the level of (40 per cent) or caudad to (48 per cent) the superior segment artery, never cephalad to it.

In 8 per cent a lingular artery arose in very close relation to lower lobe segmental arteries. In a total of 40 per cent, a lingular artery was a branch of a bifurcate, trifurcate, or quintuplate pattern which served the upper lobe (anterior or posterior segments) and/or lower lobe segments.

A lingular artery in the interlobar fissure arose at or below the origin of the superior segment artery in 88 per cent of all dissections. Therefore in all these instances, in left *lower* lobectomy, the superior segment artery would require separate ligation, to be followed by ligation of the basal segments artery or arteries below the interlobar origin of the lingular artery.

In 84 per cent, an anterior segment artery arose, near the hilus anteriorly, directly from the left pulmonary artery or indirectly from another artery. The anterior segment was supplied by more than one artery in 76 per cent.

The first branch of the pulmonary artery was exclusively for the anterior segment in 52 per cent. In the remaining 48 per cent, there were seven patterns, varying from 4 to 12 per cent each, in which the first branch of the pulmonary artery gave branches to the following segments or combination of segments: anterior-apical, apical, lingula, anterior-lingula, posterior, anterior-posterior, and anterior-lingula-posterior.

The apicoposterior segments were respectively supplied solely by a common trunk or two separate trunks direct from the left pulmonary artery as follows: common trunk, 16 per cent; two trunks, 40 per cent (combined, 56 per cent). Other highly variable patterns comprised the remaining 44 per cent. In one instance, the posterior segment was supplied by one artery arising at the superior level and two arteries arising in the interlobar fissure.

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A MODIFIED STERNAL RETRACTOR FOR BILATERAL ANTERIOR THORACOTOMY

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RECENT developments in open intracardiac surgery, utilizing general body hypothermia or temporary extracorporeal circulation, require extensive exposure of the heart and great vessels. Bilateral anterior thoracotomy through the third, fourth, or fifth intercostal spaces with transverse division of the sternum provides optimum access to the heart under these circumstances and is widely accepted as the incision of choice. When this incision is used,

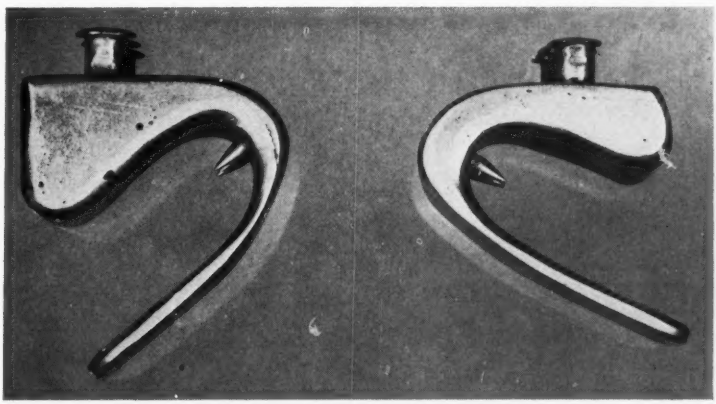

Fig. 1.—Photograph of stainless steel retractor blades for spreading the transversely divided sternum. The blade shown on the left which engages the cephalad fragment is angled at 35 degrees and the one on the right for the lower fragment is at a 25 degree angle.

retraction of the ribs by conventional rib spreaders is often unsatisfactory for a number of reasons. As the chest is sprung open, the ribs may lose their parallel alignment resulting in slipping or dislodgment of the spreader. Furthermore, such retractors may depress the lower segment of the divided sternum leading to impingement upon the heart. Even when two retractors are used, one being inserted in each end of the incision, the result leaves much to be desired. Retraction of the sternum by assistants using rake retractors may provide excellent exposure, but for obvious reasons self-retaining retractors are more desirable.

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In order to meet the requirements for optimum retraction in bilateral anterior thoracotomy, a set of detachable retractor blades of stainless steel was made for use with the Burford-Finochietto self-retaining rib spreader (Fig. 1). These sternal blades are attached to the spreader arms in the conventional manner (Fig. 2). The blade which is applied to the cephalad sternal fragment is notched at an angle of 35 degrees, and the lower blade is angled at 25 degrees. Thus, the retraction is maintained with the sternum opened outward so that impingement upon the heart is prevented. Two prongs in the notch of the angled blades engage the soft marrow cavity of the sternum providing additional stability.

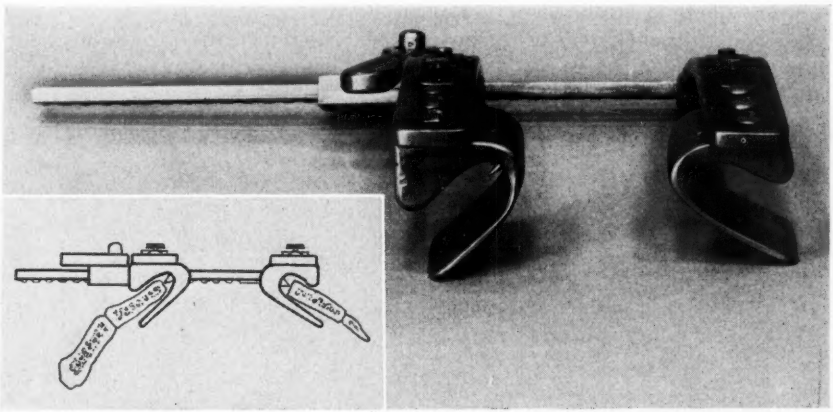

Fig. 2.—Photograph showing blades attached to standard Burford-Finochietto self-retained rachet-type retractor. Drawing in insert shows outward displacement of sternal fragments by the retractor.

Although we have used this retractor only in large children or adults, a similar device could be adapted for infants and small children. In approximately 25 patients operated upon through bilateral thoracotomy for cardiac defects under hypothermia or cardiac by-pass, this retractor has been uniformly satisfactory.

AN INSTRUMENT TABLE FOR CARDIAC SURGERY

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APPROXIMATELY 250 intracardiac procedures have been performed at the University of Minnesota Hospitals since April, 1954, using a specially designed instrument table.† This device has the advantage that the nurse can easily anticipate the surgeon's needs since with it in use she has a complete view of the operative field and can reach most instruments without turning to the backtable. Moreover, the critical period of cardiac by-pass is not needlessly

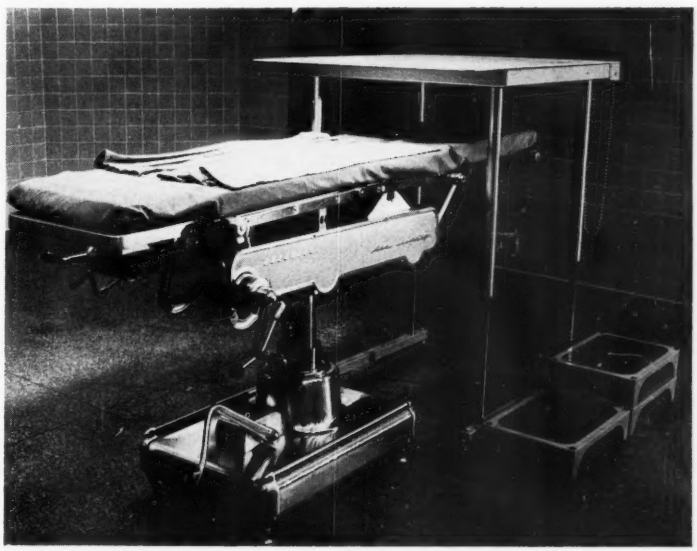

Fig. 1.—The instrument table in position showing its adjustability and accompanying foot stands.

lengthened by unnecessarily wasteful activity on the part of the instrument nurse. The table can be prepared preoperatively and, after the patient is draped, moved into a position straddling the operating table. It can easily be moved out of the way, if this is required, or elevated to facilitate possible

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†Manufactured by Phelan Manufacturing Company, Minneapolis, Minn.

emergency maneuvers. At the close of the operative procedure, the table can be removed intact without contaminating the instruments and area. Should subsequent reopening of the wound prove necessary, the table can be moved quickly back into position.

Fig. 2.—Table in use showing the availability of instruments during surgery.

The table has an adjustable height ranging from 37½ to 60 inches. It is raised or lowered by means of a bead-chain drive. The table top measures 34 by 42 inches and affords ample space for the instruments used in almost any type of operative procedure. Specially designed step stands have been devised for this table which allow the instrument nurses to work at any desired height. They are conductive and lock, either end-to-end or stacked, and provide stability for the standee.

Although this table was originally designed for intracardiac procedures, obviously its advantages are applicable to other operations.

Announcements

THE AMERICAN ASSOCIATION FOR THORACIC SURGERY

Notice of Annual Meeting

The Thirty-eighth Annual Meeting of the American Association for Thoracic Surgery will be held May 16, 17, and 18, 1958, in Boston, Massachusetts. Headquarters will be at the Hotel Statler.

Requests for Hotel Reservations

These should be mailed directly to Mr. F. Whitehouse, Front Office Manager, Hotel Statler, Park Square, Boston, Massachusetts. Please mention this Association, the type of accommodation desired, the date, and approximate hour of arrival and departure. If accommodations are desired elsewhere in Boston, please communicate directly with the hotel of your choice.

Thoracic Surgery Forum

The Thoracic Surgery Forum will be continued as a special session at the 1958 meeting. The Forum is primarily for the presentation of short papers on current thoracic research, experimental problems, anatomical studies, and modified or new surgical techniques. The inclusion of any considerable amount of clinical material in these reports makes them unsuited for this session.

Papers at this session will be limited to ten minutes, including the showing of lantern slides or other illustrative material. At the discretion of the President, three minutes may be allowed as the maximum discussion for each paper.

All Thoracic Surgery Forum papers will be published together in one issue of the JOURNAL. Maximum length: 3,000 words without illustrations. If illustrated, an appropriate number of words must be subtracted for each cut used.

Abstracts of Papers

Abstracts of all papers for presentation at the 1958 meeting must be received on or before Dec. 15, 1957, otherwise they will not be considered by the Program Committee.

Abstracts should be labeled "For Thoracic Surgery Forum," or "For Regular Program." They should contain from 200 to 250 words and accurately reflect the content of the completed paper.

Six, repeat, six clearly legible copies of *each* abstract must be sent to Miss Ada Hanvey, Administrative Assistant, The American Association for Thoracic Surgery, 600 South Kingshighway, St. Louis 10, Missouri.

Essayists selected for the program are reminded that papers presented at the meeting *must* be handed to the secretary immediately after their presentation. If they are not ready at that time there is a chance that they will not be published in THE JOURNAL OF THORACIC SURGERY.

Motion Picture Session

Provisions have been made to show a limited number of motion pictures to be selected on a competitive basis. *The films themselves,* together with descriptive material concerning length of presentation, etc., must be received by Dr. Karl P. Klassen, The Ohio State University, Columbus 10, Ohio, before Dec. 15, 1957.

It is planned to run the motion pictures in a smaller room at the same time as the scientific sessions. If the films are well labeled, it will not be necessary for the author to be present for comment.

Applications for Membership

Applications for Associate Membership in the Association must be received by the Membership Committee not later than Dec. 15, 1957, otherwise the application will be deferred for consideration until the 1959 meeting.

Applicants must be sponsored by three Active or Senior Members of the Association. In addition to the signatures on the application form, the sponsors will forward a *separate* letter concerning the applicant directly to:

Dr. Duane Carr
Chairman of the Membership Committee
899 Madison Avenue
Memphis, Tennessee

Sponsors are reminded that *new* letters of recommendation must be forwarded to the Membership Committee should their applicants be held over for reconsideration a *second year.* The new letters of recommendation should also be sent to Dr. Carr, the Chairman of the Membership Committee.